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American Journal of Diseases of Children

VOL. 2 DECEMBER, 1911 No. 6

INFANTILE PARALYSIS

WITH ESPECIAL REFERENCE TO ITS OCCURRENCE IN MASSACHUSETTS, 1907-1910 *

ROBERT W. LOVETT, M.D., AND MARK W. RICHARDSON, M.D.
BOSTON

The problem of infantile paralysis is definitely before the physicians of the United States.

In the twenty years from 1881 to 1900 there were reported in this country 200 cases. In the five-year period beginning in 1900 and ending in 1904 there were reported 100 cases. In the five years beginning in 1905 and ending in 1909, 5,400 cases, and in one — the year 1910 — approximately 9,000 cases. Such an extraordinary increase, in which Europe has in no way shared, brings before us very seriously our obligation to investigate the disease, to take account of our stock of knowledge regarding it and to do what we properly can do to quiet the panic that exists in the minds of the laity, by placing before them what we know and by asking their assistance to help us to learn more (Charts 1, 2 and 3).

The present paper will consist of a brief résumé of our present accepted knowledge of the various aspects of the disease and an analysis of the studies in Massachusetts made by the State Board of Health for the four years beginning in 1907.

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*Read at the Los Angeles Session of the American Medical Association, at a joint session of the Section on Practice of Medicine and the Section on Diseases of Children, June , 1911.

*This paper was prepared with the co-operation of Henry P. Walcott, M.D., Chairman of the Massachusetts State Board of Health, and of Theobald Smith, M.D., Milton J. Rosenau, M.D., John Lovett Morse, M.D., and James H. Wright, M.D., Advisory Committee on Infantile Paralysis to the Massachusetts State Board of Health.

In the field work the Board has had for two years (1909-1910) the services of Dr. Philip A. E. Sheppard of Boston, and Dr. Thomas P. Hennelly, now of Pittsfield, Mass. The epidemic in the Deerfield Valley in 1908 was investigated by Dr. Herbert P. Emerson, of Springfield, then State Inspector of Health for that district. Much valuable assistance has been rendered furthermore by the other State Inspectors of Health throughout the Commonwealth.

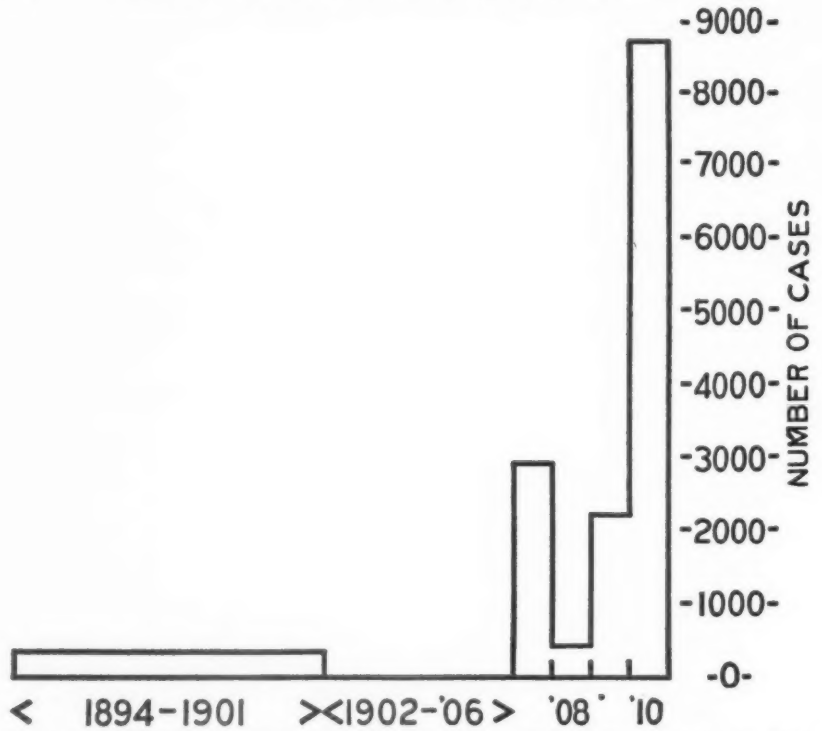

Chart 1.—Prevalence of infantile paralysis in the United States, 1894 to 1910.

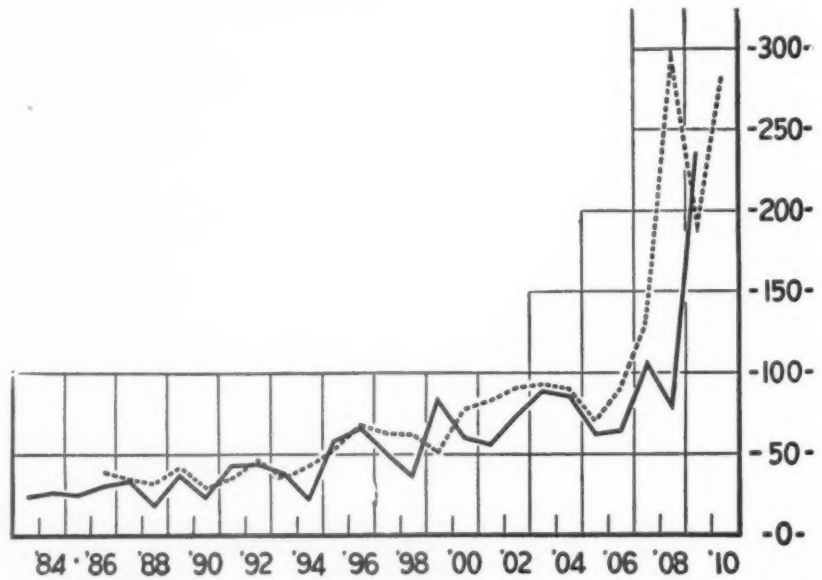

Chart 2.—Number of new patients with infantile paralysis treated at Children's Hospital, Boston, indicated by the solid black line; those treated at the New York Orthopedic Dispensary and Hospital by the dotted line; both by years.

I. RECENT HISTORY AND ETIOLOGY OF INFANTILE PARALYSIS RÉSUMÉ OF PRESENT ACCEPTED KNOWLEDGE

History.—The disease is not a new one. Although the first clear description of it was given in 1840 by Heine, a German, it was fairly well described by an English physician, Michael Underwood, in 1774. Since the perfectly clear description of the disease by Heine, in 1840, successive additions to our knowledge have been made, the most important contributions until very recently having been by Norwegian and Swedish authors, who had unusual opportunities for the study of the disease between 1899 and 1907.

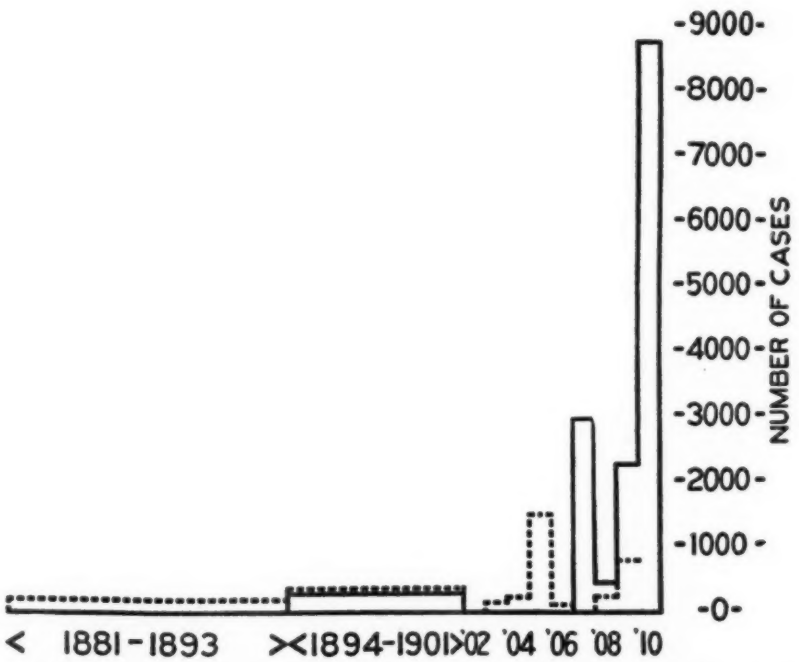

Chart 3.—Relative prevalence of infantile paralysis in the United States and Europe and Australia; the solid black line refers to the United States; the dotted line to the following countries: Italy, Sweden, Norway, Germany, Austria, France, England and Australia.

Infectious Character.—Although it had long been suspected to be an infectious disease, this fact was not absolutely proved until November, 1909, when Flexner and Lewis of the Rockefeller Institute in New York demonstrated, by inoculation of a series of monkeys with tissues from children dying of the disease, that the process was an infectious one, placing it in the category with rabies, yellow fever and foot-and-mouth disease. This discovery was undoubtedly the most important addition to our knowledge of the disease ever made.

Epidemic Character.—That the disease was infectious was suggested by the fact, recognized as early as 1881, that it occurred in epidemics. Indeed, a questionable epidemic was reported in 1841 by a Louisiana physician. From 1881 until the present time, epidemics have been reported with increasing frequency, and since 1905 the size of epidemics has largely increased. We must remember, however, that of late greater attention has been paid to the disease and a larger number of cases has been recognized, so that the increase is in a measure apparent. Nevertheless, the increase has been too great to be accounted for by any theory of increased attention to the disease.

Epidemic History.—The first great epidemic of modern times occurred in Norway and Sweden in 1905, 2,000 cases being reported in Norway and Sweden together. In 1907 the first great American epidemic occurred, 2,500 cases being reported in and about New York, since which time the disease has been increasing irregularly in the United States, making a very great increase in 1910 over any previous record.

Infantile paralysis first appeared in the United States to any considerable extent in 1894, when Caverly of Rutland reported 132 cases in the Otter Creek Valley, Vermont. Some ten small epidemics were reported in the United States during the next twelve years, and in 1907 the largest epidemic on record, that in New York, occurred, some 2,900 cases having been reported in that year. In 1908 the disease was apparently quiescent, only 392 cases being reported in the United States. This was followed by outbreaks in 1909 in various parts of the country, and in Cuba for the first time, with a total for the year of 2,343 cases in all.

In 1910 the disease assumed much more serious proportions and was present in forty-three states. Several states had epidemics, 500 or more cases occurring in the District of Columbia, Iowa, Massachusetts, Minnesota, Indiana and Pennsylvania, and from 200 to 500 in Kansas, Maryland, New Hampshire, New York, Rhode Island, Virginia, Washington and Wisconsin (Chart 4).

The reasons for this marked increase in the occurrence of infantile paralysis are not clear, but certain considerations are of interest and possibly of importance.

Question of Importation.—No explanation has been given for the sudden increase of the disease in Norway and Sweden in 1905, but after that epidemic it seems not unreasonable to suppose that the disease may have been increased in this country by Scandinavian immigrants, who arrived to the number of about 42,000 in the year 1907. The epidemic of 1907 in New York may have had its origin in this way, and the disease may have been carried in a similar manner to the middle west, but the explanation is not wholly satisfactory, inasmuch as the outbreak of the disease in the west did not conform closely to the distribution of Scandi-

BRITISH COLUMBIA
75
ALBERTA
11
NEWFOUNDLAND
1
WASHINGTON
300
MONTANA
170 (EST.)
NORTH DAKOTA
150
ONTARIO
179
QUEBEC
PRESENT
OREGON
112
IDAHO
96(F)
WYOMING
RARE
SOUTH DAKOTA
86
MINNESOTA
1000
WISCONSIN
370 (EST.)
MICHIGAN
43
NEW YORK
225
NEW BRUNS
FEW
NOVA SCOTIA
PRES.
MAINE
210 (EST.)
48
845
R.I. 231(F)
168
70(09-10)
DEL. 8
MD. 300(EST.)
CALIFORNIA
NEVADA
9
(09-10)
UTAH
120(EST.)
COLORADO
9 (F)
NEBRASKA
50
IOWA
651
ILLINOIS
137 (F)
INDIANA
500 (EST.)
OHIO
52(F)
PENNSYLVANIA
1000+
W. VIRGINIA
500
RARE
VIRGINIA
350
KENTUCKY
FEW
TENNESSEE
NONE KNOWN
N. CAROLINA
FEW
S. CAROLINA
85(F)
GEORGIA
FEW
ALABAMA
FEW
MISSISSIPPI
RARE
LOUISIANA
50±
FLORIDA
19
KANSAS
198
MISSOURI
174
OKLAHOMA
20
INDIAN TERR.
ARKANSAS
NO. INF.
ARIZONA
30 (EST.)
NEW MEXICO
NONE KNOWN
TEXAS
FEW
HAWAII
33
100
LEGEND
DISEASE REPORTABLE BY LAW. NOT REPORTABLE IN OTHER STATES.
(F)- FROM A SERIES BY FROST
(EST.)- NUMBER OF CASES ESTIMATED
± - APPROXIMATE.
CANAL ZONE
1 (WHITE)
PORTO RICO
ABSENT
DISTRIBUTION OF
INFANTILE PARALYSIS
IN UNITED STATES
IN 1910

Chart 4

navian immigrants. The Massachusetts epidemic can be reasonably explained on the ground that it came from New York. In fact, the New York epidemic was sufficiently large to be a possible source for the outbreaks in various parts of this country.

Travel.—It has been noted in the distribution of all epidemics that they tend to spread along the lines of greatest travel; certain figures were, therefore, analyzed with regard to passenger traffic in the United States.

Steam Railways.—In 1904 there were 715,400,000 passengers carried by the railroads, and in 1908 there were 890,000,000, the average number of passengers per train mile being in the first period forty-six, and in the second fifty-four; that is, there were 175,000,000 more passengers carried per year in 1908 than in 1904.

Electric Railways.—The only available figures with regard to electric railway traffic are those for 1902 and 1907. In 1902 the number of passengers carried was 5,536,000,000, and in 1907 it was 9,500,000,000, an increase in round numbers of 4,000,000,000, or about 75 per cent. in five years.

Automobiles.—Another important factor as regards increased travel is the automobile, and here the only figures which it has been possible to analyze are those relating to Massachusetts. In 1904 there were 4,000 automobiles registered in Massachusetts; in 1909, 24,000; that is, in the five-year period the number of automobiles increased six times.

Conclusions from Study of Travel.—These figures show a very decided increase in travel in the United States in five years, an increase wholly out of proportion to the increase in population. It is not at all unlikely that it is one factor by which the disease has been distributed over the country; and if this conclusion is applied to the year 1894 when the Rutland epidemic occurred, the increased communication by travel becomes very much more marked by comparison.

Having considered these general aspects of the disease we now come to the closer consideration of its etiology.

ETIOLOGY

The disease is an infectious one, caused by an ultramicroscopic virus which passes through the closest porcelain filters, its infectious character having been proved by the fact that it can be propagated in a second and many subsequent series of monkeys from tissues of a first series.

The virus is contained in the brain and spinal cord, the mucous membrane of the nasopharynx, in infected lymph-nodes, in the salivary glands, and, in the acute stage, in the cerebrospinal fluid and the blood.

The disease may be produced experimentally in monkeys by intracerebral, intraperitoneal, subdural, intraneural, perineural and subcutaneous injections, and by implantation in the anterior chamber of the

eye. The disease has been caused also by the introduction of the virus into the stomach and intestines. Successful inoculation has been accomplished by rubbing the virus into a scarified or even a sound nasal membrane, and two observers succeeded in producing the disease by inhalation of an emulsion containing the virus and by implantation in the trachea. All investigators agree, however, that the intracranial route is the best for inoculation.

Inoculation of horses, calves, goats, pigs, doves, sheep, rats, cats, mice, chickens, guinea-pigs and dogs have proved negative, and monkeys have proved the most satisfactory animals for study. A paralysis may be experimentally produced in certain varieties of young rabbits and may be transmitted from one generation to another of rabbits and finally back to monkeys by successive inoculations.

The virulence of a suspension is not impaired by drying (to nine days), by freezing, nor by suspension in glycerin for five months, but it is killed by exposure to a temperature of from 45 to 50 C. in half an hour. It is also killed by such weak disinfectants as 1 per cent. solution of peroxid of hydrogen (perhydrol of Merck) by 1 to 500 solution of permanganate of potash, and by a powder containing menthol 0.5, salol 5, and boric acid 20.

The organism is not visible with the highest powers of the microscope and will not grow on the culture media in general use.

The disease in monkeys is very much like that in children, but is much more fatal; in fact, so large a proportion of inoculated monkeys die that the study of the affection clinically in them is difficult. No instances as yet have been reported in which one monkey has taken the disease from another, although long continued and intimate contact has been maintained.

The incubation period in monkeys is from five to forty-six days, with an average of eight or nine days.

IMMUNITY

A child or animal that has once had the disease becomes apparently immune and the blood of children and monkeys recovered from the disease has been found to render other monkeys refractory to subsequent inoculation, the specific antibodies persisting in the blood for three years in one case, but being absent in another case eleven years after the onset.[1] This fact, of course, suggests that a preventive serum may be hoped for, but as yet the serum treatment is wholly in an experimental stage, and not yet ready for use in cases of the human disease.

1. Netter and Levaditi: Comptes rend. Soc. de biol., 1910, lxviii, 855.

PERSONAL TRANSMISSION — DIRECT AND INDIRECT

In discussing the question of the transmission from patient to patient, the use of the technical and restricted term "contagious" will be avoided and the term "transmissible" will be employed.

The disease may be transmitted apparently in three ways: (1) By direct contact of a healthy child with a sick one; (2) by indirect contact by means of healthy persons passing from the sick to the well; and (3) in certain very rare instances a child has become ill shortly after occupying a house from which an affected family had moved.

Comparative Transmissibility.—The disease is, however, apparently less transmissible than other so-called contagious diseases with which we are more familiar.

In Minnesota a very careful investigation was conducted by Hill as to the relative transmissibility of infantile paralysis compared with that of scarlet fever, measles, etc., in the same territory. Of persons known to have been exposed to diphtheria, scarlet fever and infantile paralysis, the percentage contracting the disease was as follows: Scarlet fever, 22 per cent.; diphtheria, 17 per cent.; infantile paralysis, 6 per cent. Taking the instances of these same diseases, where one case existed in a family, the number of other cases occurring in the same family was as follows: Scarlet fever, 40 per cent.; typhoid fever, 30 per cent.; diphtheria, 29 per cent.; infantile paralysis, 17 per cent. The disease is thus apparently less transmissible than scarlet fever, typhoid fever or diphtheria.

The same evidences of mild transmissibility were shown in an investigation by Emerson, of an epidemic in the Deerfield Valley in Massachusetts in 1908, where a rural community offered excellent opportunities for study of contact. Furthermore, such instances as the following are difficult to explain by any theory of personal contact, direct or indirect:

In a sporting camp in a northern forest the 5-year old son of the proprietor had been in the camp since early spring. He had never left the camp during the summer. The camp was 5 miles from any other camp or house and 10 miles from the frontier town. There had been no other children in the camp during the summer and no illness of any kind in the guests or among animals. The few guests who were there came from the large cities, put on their hunting and fishing clothes and lived out of doors. In October the child developed a typical infantile paralysis, which still persists in one leg.

Evidence of transmission of the disease by personal contact from other epidemics is to be found in Wickman's Study, Shidler's report,[2] an epidemic observed by Frost,[3] and the Massachusetts studies.

2. Shidler: Pediatrics, 1910, xxii, 539.
3. Frost: Public Health Bulletin 44.

Possible Factors Other Than Personal Transmission.—We come next to the consideration of the possibility of fruit, dust, insects, and animal paralysis as factors in the causation of infantile paralysis.

Fruit.—As to fruit, the high cost of living has undoubtedly led to a diminished consumption of meat, and presumably to an increased consumption of fruit. Traffic in fruit has increased steadily between 1900 and 1908, the increase being from one to two million tons a year, up to 1905, since which time there has been no such sharp increase. So far as these figures go, therefore, it would not seem that any very striking factor was to be found in this analysis. Common observation would, however, seem to show that in Massachusetts, at least, there are more fruit stores and apparently more fruit in use than was the case five years ago.

Dust.—In order to determine the rôle of dust in the etiology of infantile paralysis, the rainfall of the country was analyzed for two periods: first, the five years from 1900 to 1904; and second, the five-year period from 1905 to 1909, when we had the greater prevalence of the disease. The total rainfall during the last five years on the Atlantic seaboard has shown everywhere a diminution, reaching a high figure in New England. On the contrary, the regions in the middle west affected by the epidemic of infantile paralysis in 1909 showed an increased rainfall during the last five years, the rainfall being as much in excess of the normal as in New England it was deficient. In the middle Atlantic states, however, including New York, the rainfall in the two periods was practically the same. Excessive dryness in the last five-year period cannot, therefore, be put down as a condition pertaining to the affected regions, if one considers New England along with Nebraska and Minnesota. But we must remember that the germ is not injured by freezing or drying, and that the season of occurrence is certainly the season of greatest occurrence of dust. Moreover, a German observer found the disease in an unduly large proportion of shoemakers' children who were much in the shops where the dried dust was flying about, and the distribution of the disease along lines of travel suggests the possibility of such a mode of transmission.

Insects.—There are certain facts connected with the disease which suggest the possibility of its being transmitted by insects. This matter will be dealt with in speaking of the Massachusetts studies.

It has been shown experimentally by Flexner that if flies are allowed to feed on the cord of a monkey dying of the disease that they may carry the virus for forty-eight hours.[4]

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4. Flexner, Simon and Clark, Paul F.: Jour. Am. Med. Assn., June 10, 1911, p. 1717.

Domestic Animals.—With regard to paralysis in the domestic animals, cases of paralysis strikingly suggestive are reported in horses, pigs, cats, dogs, rabbits, chickens, and other animals. A study of the distribution of animal paralysis in Massachusetts by means of letters addressed to veterinarians and animal inspectors showed, however, that in 1909 the distribution of cases of animal paralysis so reported did not in any way correspond to the distribution of the human disease for the same year, but much more extensive investigation along this line is necessary.

The conclusion must be that, in a consideration of etiology, infantile paralysis cannot be dismissed as a disease due only to transmission from patient to patient, but that these other factors must also be considered as possibly important.

Season of Occurrence.—In considering the etiology of the disease one is struck with the remarkably uniform seasonal occurrence which prevails in the north or in the tropics, in Europe or America. The disease begins to appear in the early summer, reaches its height generally in July, August or September, and diminishes through October, November and December, nearly to disappear in late winter and spring. In the southern hemisphere this behavior is reversed and the disease prevails most there in February and March, which is, of course, late summer and early fall in that locality.

Age.—The age of selection of the disease is between 2 and 3. Adult cases are comparatively uncommon, and occur chiefly in young adults. Dividing the ages into five-year periods, the following table was constructed by Frost:

TABLE 1.—AGE INCIDENCE IN FIVE-YEAR PERIODS EXPRESSED IN PERCENTAGES

Age Years	N. Y. Commission (729 Cases) Per cent. of Total	Lovett (Mass.) (615 Cases) Per cent of Total	Hill (Minn.) (325 Cases) Per cent. of Total
Under 1	8.5	7.2	6.5
1 to 5	82.0	64.5	48.6
6 to 10	6.4	15.9	23.7
11 to 15	1.9	5.0	7.7
16 to 20	0.68	2.4	6.5
Over 20	0.40	5.0	7.0

General Incidence.—It affects all classes, rich and poor, in the cities and the country, in the tenements and in detached houses, on the top floor of houses, on the ground floor and in basements.

II. PATHOLOGY, SYMPTOMS, DIAGNOSIS, PROGNOSIS, PREVENTION AND TREATMENT

General Grouping of Symptoms and Types.—As a working hypothesis we may keep in mind the following as to symptoms and types of paralysis:

The infection enters the body by channels as yet unknown. In some instances this infective agent causes death from general paralysis due to extensive destruction of certain spinal and cerebral centers; in a second class of cases this agent causes some permanent loss of power in part or all of one or more limbs, the back, the abdomen, or in groups of muscles supplied by cranial nerves. In a third class, the same agent causes a very local yet probably permanent paralysis in one small muscle or small group of muscles, e. g., the extensor muscles of one little finger or certain muscles of the spine.

In a fourth class, weakness or transitory paralysis ending in recovery occurs as the result of the same infection. And lastly, in a fifth class, the infective agent causes fever, prostration and perhaps pain, but does not cause sufficient destruction to bring about any paralysis or weakness.

If we recognize this descending scale in the severity of the disease as depending probably either on the dose of the poison received or on the resistance of the individual, and recognize that the involvement of the spinal centers is perhaps after all incidental rather than essential, we may well believe that the disease is far more prevalent than is generally supposed, and especially in its abortive forms.

PATHOLOGIC ANATOMY AND HISTOLOGY

The characteristic lesions of the disease are found in the spinal cord and are most marked in the anterior horns of the gray matter of the cervical and lumbar enlargements.

In the gray matter in the early stages of the lesions are

1. Congestion of blood-vessels.

2. Edematous softening, sometimes accompanied by hemorrhage.

3. Infiltration with cells. (The cells diffusely infiltrate as well as aggregate together in foci and about the blood-vessels. They are of various kinds, but are chiefly lymphocytes and larger cells whose origin is not generally agreed on, and which are sometimes phagocytic.

4. Degeneration and diminution in the number of nerve-cells. The cytoplasm of the nerve-cell becomes hyaline and disappears leaving the nucleus naked and necrotic, and it also eventually disappears. The degenerated cell may be surrounded and invaded by the infiltrating cells. All of the nerve-cells in a given area do not always show degenerative changes, and more of them may be involved in one side of the cord than in the other. In the later stages of the disease, in extensively affected regions, nerve-cells and their processes are absent, the tissue is disintegrated and there is infiltration with phagocytic cells, plasma cells, proliferated glia cells, and connective tissue cells. Eventually, neuroglia with some connective tissue replaces the disorganized tissue and the part may appear atrophied to the naked eye.

In the white matter in the early stages of the disease, the lesions consist of congestion, edema and cellular infiltration of the adventitial lymph spaces of the blood-vessels and to a slight extent of the white matter as well. The infiltrating cells are chiefly lymphocyte-like cells. Small foci of these cells have been noted in the white matter. In some cases, acute degenerative changes have been observd in the anterior roots.

The pia mater also shows some infiltration, chiefly with lymphocytes, which is greatest in the anterior portions of the cord in the lumbar and sacral regions.

In the spinal ganglia there may be the same infiltration as in the white matter, without marked changes in the ganglion cells.

In the later stages of the disease there is degeneration and atrophy of nerve fiber tracts, anterior roots, motor nerves and muscles, depending in extent and location on the nerve-cells which have been destroyed.

In the medulla and pons similar lesions to those in the cord are found, but the degeneration of the ganglion cells is not so extensive as in the cord.

The brain as a rule shows comparatively slight lesions. Congestion of blood-vessels, small hemorrhages, cellular infiltration of the vessel sheaths and of the brain substance in foci have been observed in various situations. In old cases, foci of sclerosis have been observed.

In acute cases, the thoracic and abdominal organs show changes which may be observed in other acute infectious diseases. A hyperplasia of the lymph-adenoid tissue of the intestine, mesentery and spleen is commonly observed in addition to acute degeneration of the myocardium, liver and kidneys, and there may be bronchopneumonia.

There is nothing in the pathologic anatomy and histology of the disease which shows the mode of entrance of the infectious agent into the body nor whether it reaches the central nervous system by the blood or by the lymph-stream.

SYMPTOMATOLOGY OF INFANTILE PARALYSIS

It is impossible in the time at our disposal to give more than the most cursory description of the symptomatology of this disease. If we think of it as a poliomyelo-encephalitis with meningeal complications and appreciate the fact that the disease may affect any or all parts of the gray matter of the central nervous system, and in all possible combinations, it is easier to understand the innumerable manifestations of the disease and it is less likely to be overlooked and mistaken for other conditions. The best classification of the various manifestations of the disease is that of Wickman, which is as follows:

1. Ordinary spinal paralysis; anterior poliomyelitis.

2. Progressive paralysis, usually ascending, less often descending; Landry's paralysis.

3. Bulbar paralysis; polio-encephalitis of pons.

4. Acute encephalitis; giving spastic mono- or hemiplegia.
5. Ataxic type.
6. Meningitic type.
7. Polyneuritic; multiple neuritic type.
8. Abortive type.

Initial Symptoms.—It will be well, perhaps, before taking up the special clinical types of this disease to consider briefly its initial symptoms and the possibilities of making a diagnosis before the appearance of the paralysis. There is, in the vast majority of cases, nothing characteristic about the onset. The symptoms are, in general, those of an acute infection. In many instances, however, gastro-intestinal symptoms predominate, while in others those referable to the respiratory tract are the most marked. Sweating, marked nervous irritability and general hyperesthesia are present in many instances before the onset of the paralysis, but they are not at all constant. Their presence points strongly toward this disease but their absence does not count against it. It is safe to say, therefore, that at present there is nothing about the early symptoms to justify a positive diagnosis of infantile paralysis before the onset of the paralysis, although the appearance of the symptoms of an acute infection in a child, known to have been exposed to this disease or during an epidemic, especially if accompanied by sweating, nervous irritability and general hyperesthesia, are sufficient to justify a probable diagnosis.

A leukopenia has been found in animals during the late prodromal and early part of the acute stage. It is, however, not a constant symptom in man and is often replaced by a hyperleukocytosis. It is possible that there is a lymphocytosis in the early stages, but the evidence at present is not sufficient to show whether it is at all constant. It is possible that both the absolute and differential count of the leukocytes may prove useful in the early diagnosis of infantile paralysis. At present, however, they are of little or no assistance.

Experimentally, there is an increase in the number of cells in the cerebrospinal fluid during the prodromal stage, the polynuclear cells at this stage outnumbering the mononuclear. After the appearance of the paralysis, however, the mononuclear cells quickly exceed the polynuclear. A fibrin clot is also often formed during the prodromal or early part of the acute stage. The cerebrospinal fluid, in the only reported instance in which it has been examined in man during the preparalytic stage, was slightly opalescent, gave a marked protein reaction with Noguchi's test and showed an excess of polynuclear cells. The formula had begun to change to the lymphocytic, however, before the appearance of the paralysis. It seems safe to say, therefore, that in most instances a positive diagnosis of infantile paralysis can be made in the preparalytic stage by means of lumbar puncture, and that this is the only way in

which a positive diagnosis can be made at this time. The value of this procedure is considerably limited, however, by the variability of the early symptoms and the consequent lack of anything to definitely suggest its use. In order to recognize the disease early, therefore, before the onset of the paralysis, it would be necessary to do a lumbar puncture on every sick child, which hardly seems a rational procedure.

Abortive Type.—What has been said as to the symptomatology and diagnosis of the preparalytic stage of infantile paralysis applies equally well to the abortive type (Type 8 under Wickham's classification), meaning by this term the type in which there is never any paralysis. There has been some doubt as to the existence of this type in the past, because there was no way of positively proving that the cases supposed to be of this type really were examples of this disease at all, the evidence being merely presumptive. Flexner and Clark and Frost have, however, recently demonstrated the presence of specific immune bodies in the blood of persons supposed to have had abortive attacks of infantile paralysis. The blood serum of these patients inactivated the virus of the disease, so that the infection of monkeys was impossible. There is no doubt, therefore, that abortive cases do occur. It is probable that they are as common as, if not more common than, those with paralysis. The importance of the recognition of these cases, if the present conception as to the rôle of "carriers" in the spread of the disease is correct, is obvious.

1. Ordinary Spinal Type.—It hardly seems necessary to take up in detail the symptomatology of this type, with which everyone is familiar. The paralysis reaches its maximum in from a few hours to three or four days, is followed by a stationary period of from one to four weeks, which is in turn followed by a period of improvement, lasting from six months to a year, the final paralysis always being less than the initial. It is impossible to prophesy in the acute stage how extensive the final paralysis will be. In general, however, the final paralysis varies directly with the initial. The final paralysis is always flaccid; its distribution usually monoplegic or hemiplegic. In most instances some of the muscles of the paralyzed extremity escape; not infrequently only one or two are involved. The reflexes are diminished or absent, sensation is only exceptionally impaired. Deformities develop as the result of contractures of the non-paralyzed muscles.

It is not very uncommon for other muscles than those of the extremities to be involved, while these escape. Such muscles are most often the erectors of the spine and the abdominal muscles. The involvement is, moreover, not infrequently unilateral. The extensor muscles of the neck, those of one side of the spine or of one side of the abdomen may, for example, be alone involved. The deformities resulting from the paralysis or paresis of these muscles are often misunderstood and attributed to other causes.

2. Progressive Type.—In this type the paralysis usually appears first in the legs, and gradually extends upward. In rare instances it appears first in the arms, extends downward and finally upward to the muscles supplied by the medulla. It is probable that the great majority of the cases that have in the past been described under the term of "Landry's paralysis" really were examples of this type of infantile paralysis. When the paralysis reaches the external muscles of respiration, as it very often does, death is practically inevitable and usually occurs on the third or fourth day. The diaphragm is also sometimes involved. Death occurs sooner, of course, when this happens. This form must not be confused with that in which death results from respiratory paralysis due to involvement of the centers of respiration in the medulla.

3. The Bulbar Type.—Cases of this type have in the past been usually designated as polio-encephalitis superior or inferior, according to which of the cranial nerve nuclei were involved. The nuclei may be affected singly or in all possible combinations. The symptoms depend, of course, on the nuclei involved. The facial and abducens nerves are perhaps the most often affected. There may be paralysis of deglutition and of the muscles of the larynx. When the vagus is involved there are disturbances of respiration and of the cardiac action. In such instances, the respiration is often of the Cheyne-Stokes type and the prognosis is practically hopeless. Involvement of one or more of the cranial nerve nuclei is not very uncommon in connection with the ordinary spinal type of infantile paralysis. When this happens the resulting picture is a combination of the two types.

4. Acute Encephalitic Type.—This type was described by Strümpell many years ago under the term, "acute encephalitis of children," but it has only recently been recognized as a variety of the disease under consideration. The symptoms resemble those of acute meningitis, the deep reflexes are, as a rule, exaggerated and the paralysis is spastic. The diagnosis is usually impossible without lumbar puncture. The prognosis as to life is better than it would appear and that as to recovery from the paralysis much better than in paralysis due to other cerebral diseases.

5. The Ataxic Type.—Ataxia is a prominent symptom in a certain number of cases. In a few, it is the only nervous symptom; in others, it is associated with paralysis of the cranial nerves and sometimes with a small amount of spinal paralysis. The ataxia is often distinctly of the cerebellar type.

6. The Meningitic Type.—Symptoms of meningeal irritation are not at all uncommon in the early stags of all types of infantile paralysis. These are so marked in many instances that they present the typical picture of meningitis. Headache, rigidity of the neck and back, vomiting, tonic and clonic spasms, strabismus, Kernig's sign, delirium, coma and

other signs of meningeal irritation may be present in any and all combinations. In such cases the diagnosis on the symptomatology alone is impossible before the appearance of the flaccid paralysis. Even then it is open to doubt, because flaccid paralysis is not very uncommon in meningitis, especially if it is of the tuberculous type.

A positive diagnosis can only be made in most instances by lumbar puncture. The characteristics of the cerebrospinal fluid during the preparalytic stage have already been mentioned. During the acute stage it is clear, not infrequently under somewhat increased pressure, often forms a fibrin clot on standing and always contains an excess of protein and of cells. The cells are chiefly of the mononuclear type, most of them being lymphocytes. These characteristics are, however, the same as those found in tuberculous meningitis, the type of meningitis with which infantile paralysis is most likely to be confused. A positive diagnosis between them can only be made, therefore, by the examination of the cerebrospinal fluid for tubercle bacilli. The characteristics of the cerebrospinal fluid in meningococcal, influenzal and pneumococcal meningitis are so different from those in infantile paralysis that no confusion with these conditions is possible.

The Polyneuritic Type.—Pain is a prominent symptom in many instances. It is sometimes located in the joints, but is more often along nerve-trunks or indefinite in its distribution. It is usually most marked in the paralyzed parts. There is occasionally tenderness over the nerve-trunks. The pain and tenderness are sometimes marked enough to cause the paralysis to be entirely overlooked and a diagnosis of rheumatism or scurvy to be made.

The extremities are often held rigidly and all motion as vigorously resisted as possible, because of the pain on motion. This combination of rigidity and resistance is possible, of course, only when the muscles are partially paralyzed or when some of them are intact. The failure to appreciate the significance of this combination of flaccidity and spasticity not infrequently leads to errors in diagnosis during the acute stage.

PREVENTION

The question of the prevention of the disease is of great importance, and you are probably asked as frequently as we are in Massachusetts: "What shall I do to protect my children from the disease?" — a question which we must answer fairly with due regard to the interests of the patient, remembering, however, that advice too radical will increase the panic which is sure to appear in a locality where the disease is prevalent. It seems proper to advise people with children that, other things being equal, it is wise not to go for the summer to a locality where the disease is known to be prevalent. If, however, people find themselves in such a locality, the children and members of the family should keep away from

other children who are in the acute stage of paralysis, and from children who are sick with other indefinite ailments.

In such localities it would seem wise to keep children away from children's entertainments, circuses and electric cars, and to prohibit playing with sick or paralyzed domestic animals.

Length of Quarantine.—We have at present no data as to how long the infectious period of the acute stage lasts, and any attempt to determine the length of quarantine must be mere guesswork.

With regard to the necessary length of quarantine, general consent has placed it at from two to four weeks. The recent experiments of Osgood and Lucas in this matter are, however, important inasmuch as they found an active virus in the nasopharyngeal mucous membrane of a paralyzed monkey, five and one-half months after the active stage of the disease had passed. The virus disappears from the nervous system in three or four weeks, or less, according to Flexner's experiments.

MORTALITY

The death-rate is about seven or eight per hundred, as the disease has existed in Massachusetts, but no foreign statistics show as low a figure as this, being from 10 to 22 per cent. In persons older than 10, the mortality-rate is higher than between 1 and 10, and under 1 year the disease seems more fatal.

The mortality table is interesting as showing the difference between the foreign and American death-rate.

TABLE 2.—COMPARISON OF FOREIGN AND AMERICAN DEATH-RATE

	Year	Cases	Death	Mortality Per cent.
Caverly, Vermont	1894	132	18	14.5
Wickman, Sweden	1905	868	145	16.7
Leegaard, Norway	1905	577	84	14.5
Zappert, Austria	1908	266	29	10.8
Lindner and Mally, Austria	1908	71	16	22.5
Furntrat, Steiermark	1908	433	57	13.1
Krause, Germany	1909	633	78	12.3
Muller, Germany	1909	100	16	16.0
Peiper, Germany	1909	51	6	11.7
Eichelberg, Germany	1909	34	7	20.6
Mass., U. S. A.	1907-1910	1,599	125	7.9

The mortality of cases in older children and in adults is higher than in other classes.

TABLE 3.—SHOWING HIGHER MORTALITY IN THE MORE ADVANCED AGES

	Age	Mortality, Per cent.
Wickman	12 to 32	27.6
Leegaard	15 to 30	25.8
Furntrat	Over 15	25.5
Lindner and Mally	Over 11	50.0
Mass., 1910	Over 10	20.0

PROGNOSIS

A few years ago it was supposed by most persons that practically all patients once paralyzed were to some extent paralyzed for life. But more careful study has shown us a better and more encouraging state of affairs. In the cases in Massachusetts, in 1909, a group of 150 occurring near Boston were studied in detail and it was reported that twenty-five, or 16.7 per cent., had wholly recovered inside of three months. This statement was not believed and the investigators were sent back to review their work. Each one of the children was stripped and the movements of ankle, knee, hip, shoulder, elbow, wrist and spine were examined. The statement can be definitely made, therefore, that, of this group, 16 per cent. had wholly recovered within three months.

The commonest type of final paralysis is an affection of one leg varying in degree from a slight limp in walking to a condition which requires the use of a brace; if both legs are involved, crutches are occasionally also required.

In short, a disability from infantile paralysis so great as to prevent an adult from earning his or her own living, occurs in a very small number of patients properly treated, but the ultimate condition of neglected cases is frequently distressing.

In connection with the question of treatment it is important to consider the reasons for this tendency to recovery, which are to be found in the pathology of the disease.

At the height of the disease there is an extensive perivascular infiltration of the blood-vessels of the cord, diminishing their lumen and inducing anemia and edema of the regions of the cord supplied by them. In addition to which there is also inflammatory edema of the nerve-tracts in the white matter and edema of the nerve-cells in addition to any destructive process which may be going on in neighboring cells. These changes, of course, cause loss of function in the parts affected, expressed as a temporary paralysis which passes away as the perivascular infiltration subsides. This apparently accounts for the improvement of paralysis in the early stage of the disease which exists in all non-fatal cases.

But the spontaneous improvement in the paralysis of the early history of the disease is no more important than improvement to be obtained by proper treatment in the chronic stage of the disease, after there is every reason to believe that the temporary perivascular infiltration has subsided, and the reason for this later improvement apparently rests on another pathologic basis. Assuming that the phenomena just described have ended, but that permanent destruction of certain motor centers in the spine has occurred, leaving a definite motor paralysis, the question arises, What reason is there to look for improvement in this? The answer apparently is as follows:

The spinal motor centers are grouped in cigar-shaped bundles whose length lies in the long axis of the spine. At each level of the spine are several of these bundles which overlap each other and communicate freely. Each bundle furnishes impulses to more than one muscle and each muscle receives impulses from more than one bundle. The blood-supply of the spine comes in horizontally and the lesions of this disease are most marked around the vessels. The distribution of the destructive process is therefore likely to be horizontal, while the distribution of the motor bundles is longitudinal. For this reason it is likely that if the destruction has not been too extensive, certain parts of the motor bundles will have escaped, leaving certain cells in some of them capable of function but which, in order to perform function, must acquire new connections and new groupings.

For this reason it will be seen, as will be maintained later, on the ground of practical experience, that muscle-training offers the best hope of the restoration of power to affected muscles in the chronic stage of the paralysis. It is an attempt to bring motor impulses from the brain down to the affected centers in the cord, there to establish new connections and new routes to the paralyzed muscles. The more impulses of this sort, however feeble they may be at first, to reach the affected muscles the better the chance of establishing finally routes which may serve for the transmission of impulses strong enough to secure efficient muscular contraction.

TREATMENT — ACUTE STAGE

In the matter of treatment much progress has been made in the last four years. In the acute stage meddlesome and useless treatment by stimulating drugs and counter-irritants has become largely discredited. Rest is very important, although cathartics and hexamethylenamin are of probable value, the latter drug being used with the object of setting free formaldehyd in the cerebrospinal fluid. Indeed, it has been shown experimentally that if hexamethylenamin is administered by mouth to monkeys, and if such animals are subsequently inoculated either subdurally or by the intracerebral route, paralysis is, in a certain proportion of cases, delayed or prevented. The value of the drug, therefore, in the prevention of infection would seem to be indicated, but there is as yet no experimental evidence to show that the drug is of much use after the disease has once begun.

In view of the pathologic condition, it is simply common sense to keep the patient recumbent and quiet and to avoid irritation of the muscles supplied by the affected nerve-centers. Massage and electricity should not, therefore, be used until there is reason to believe that subsidence of the inflammation has taken place. We may probably interpret the tenderness of the limbs as showing, while it lasts, the persistence of

some degree of spinal or neural inflammation and until that has wholly disappeared we are not justified in the use of passive movements, massage or electricity.

Some men of large experience discourage all active treatment before the expiration of six weeks at least, but excellent results are frequently obtained by beginning treatment earlier on cases characterized by slight and transient tenderness. We should, however, discourage all active treatment until tenderness has wholly disappeared.

Another point of primary importance in early treatment is the prevention of contractures and deformities due to unopposed muscular action. Except in paralysis of the back and shoulder these contractures and deformities are always preventable, the contracture of the tendo Achillis, e. g., which is so frequent and so troublesome, being easily prevented by the use of splints supporting the foot at a right angle.

We read much concerning the relative merits of electricity and massage in the restoration of muscular power, but who shall pronounce as to the value of either in an affection in which paralysis varies so much in extent and severity and the recovery from which is so surprisingly different, irrespective of methods of treatment? Both methods have as an object the stimulation of muscular fibers and the maintenance of muscular nutrition, but both are inferior to muscular training in active treatment. This method of treatment is most useful in connection with massage or electricity and aims to cultivate the transmission of a voluntary motor impulse from the brain, down the cord to the paralyzed, weakened or stretched muscles. A flicker of motion, in an affected muscle, is often the forerunner of a return of some degree of power, and assistance by the hand in performing voluntary movements of the foot, for example, is often of great use.

Apparatus.—As to the use of apparatus, when a child in attempting to stand cannot, for example, hold the knee extended in weight-bearing, or when he stands in a position of deformity, for instance, with flat foot, apparatus should be applied to restore normal relations. Nothing but harm can result from sitting or going about with a curved spine, a hyperextended knee, or a foot rolled over on its inner or outer border. Stretching of muscles and ligaments and permanent bony deformities which are sure to result are worse than the use of braces, objectionable as the latter are.

Contractures which exist in neglected cases are not extremely resistant early in their history and must be removed before satisfactory mechanical treatment can be carried out. Most of them can be removed if stretched by means of bed traction or by the use of a series of correcting plaster bandages or by cutting. Operative treatment, except for the minor tenotomies, should not be undertaken for two years after the attack and preferably not then in children under 10.

Surgical Treatment.—Operation.—Tendon transplantation is a most useful measure and now that it has passed through its period of extravagant claims and unjustified condemnation, it may be said, in properly selected cases and with proper technic, to produce generally satisfactory and often brilliant results.

It consists in transplanting the distal end of a healthy muscle to the periosteal insertion of a paralyzed one, the foot or knee or arm being thus rebalanced. Since we have learned to prolong tendons by silk, the range and application of the operation has increased. We plan our operation, dissect and cut off our good muscle and if it is too short lengthen it at its distal end by heavy silk, we then insert it where we like, a new and sound tendon forming around the silk. In this way peroneals may replace tibials at the ankle or *vice versa,* hamstrings may be carried forward and inserted into the tibia to serve as knee extensors when the quadriceps is paralyzed and the trapezius may be substituted at the shoulder for a paralyzed deltoid.

Arthrodesis.—As a means of stiffening and making stable useless joints, arthrodesis has been found a useful measure. This operation consists in opening such joints and removing enough cartilage to secure ankylosis, and is applicable to the hip, ankle, elbow, and shoulder in children over 12, and to the knee in adults but not to the knee in growing children.

Silk Ligaments.—For the correction of foot drop in paralyzed legs we have made use more and more of anterior silk ligaments, the idea being to prevent plantar flexion of the foot but still to allow dorsal flexion. Heavy strands of silk are sewed into the anterior surface of the tibia, carried down under the annular ligament of the ankle and sewed into the dorsal surface of one of the tarsal bones. This operation is distinctly preferable to arthrodesis.

Nerve transplantation has not yet emerged from its experimental stage.

Finally, one of the most important things that orthopedic surgeons have learned of late years is that the hope for improvement in these cases through efficient and suitable treatment need not be given up in six months or one year or ten years, but that the possibility of improvement seems to persist almost indefinitely.

III. INFANTILE PARALYSIS AS OBSERVED IN MASSACHUSETTS, 1907-1910

The investigation of infantile paralysis in the state of Massachusetts by the State Board of Health for the four years, 1907 to 1910, inclusive, shows that during that period 2,138 cases of the disease were reported to the office of the State Board of Health. These cases were distributed by years as follows:

1907	234
1908	136
1909	923
1910	845

It is apparent from these figures that the disease in Massachusetts has, in a general way, during the period mentioned, increased in frequency, and it has been shown that a larger number of towns were infected each year.

Two-Year Periodicity.—There is, moreover, in these figures a suggestion of what may be an important phenomenon, and that is the appearance of a two-year periodicity for this disease. This phenomenon is noticeable in the distribution in general. In 1905 occurred the great Scandinavian epidemic. In 1906 there were no important epidemics. In 1907 occurred the New York epidemic. In 1908 there were only a few small epidemics and none of importance reported in Europe. In 1909 occurred the Massachusetts, Minnesota and Nebraska epidemics, two large and one small German epidemics, a large outbreak in Austria and the epidemic in Cuba. In 1910 from Europe no large epidemics were reported so far as we have been able to learn. The occurrence of the disease in this country in 1910 has already been dealt with.

This two-year periodicity has been noted a number of times in our investigation; for instance, in 1907 the western part of the state was quite sharply affected with infantile paralysis, whereas in 1908 the disease was almost non-existent in that same region. Again in 1909 the disease cropped up, but in 1910 hardly any evidence of infantile paralysis was noted in that neighborhood. In 1908, furthermore, in the Turners Falls district, etc., a very marked epidemic occurred, involving sixty-nine cases in a quite sharply circumscribed locality. In 1909 but one case occurred in this whole region. So that one can prophesy, to a certain extent, at least, that a limited locality, severely affected during one year, will probably enjoy partial immunity in the succeeding year. On the other hand, many localities having the disease to a moderate extent seem to go on from year to year at about the same rate.

Relative Prevalence in Smaller Cities and Towns.—Infantile paralysis is a disease affecting by preference the smaller cities and towns. This statement rests on the observation of 2,138 cases in Massachusetts, which have been analyzed in this regard.

The incidence per 1,000 of the population was recorded for every affected city and town in the state for four years. For each year the cities and towns were then arranged in the order of greatest incidence and then the first twenty-five on the list were contrasted with the last twenty-five for each year.

The average population for the first twenty-five was then reckoned for the four years and proved to be 5,205. The average population of the

last twenty-five on the list, those where the incidence was least, was 52,674. A further study of the table shows that some such proportion as this existed in every one of the four years. That is to say, cities and towns where the disease was relatively least frequent were ten times as large on the average as those where it was most frequent. Furthermore, as a control, we tabulated the cases of scarlet fever reported in the state for the year 1910 in the same way. In the twenty-five cities and towns in which scarlet fever was most prevalent the average population was 6,446, and in the twenty-five where it was least prevalent per 1,000 of the population the average population was 7,633, practically the same, showing that scarlet fever showed no such predilection for the smaller cities and towns (Table 4).

TABLE 4.—COMPARATIVE PREVALENCE OF INFANTILE PARALYSIS AND SCARLET FEVER WITH REFERENCE TO POPULATION OF CITIES

Disease	Twenty-five Cities Where Disease was *Most* Prevalent	Twenty-five Cities Where Disease was *Least* Prevalent
Acute epidemic poliomyelitis	5,205	52,674
Scarlet fever	6,446	7,633

In seeking to analyze some of the conditions which were constant in the smaller cities and towns which did not prevail in the larger, the number of domestic animals in each city and town was looked into and the analysis contributes the information that the number of such animals is relatively very much greater in the smaller cities and towns than in the larger. Such a fact may have no significance whatever, or it may in the future prove of importance (Table 5).

TABLE 5.—PREVALENCE OF INFANTILE PARALYSIS WITH REFERENCE TO THE NUMBER OF ANIMALS IN CITIES AND TOWNS

Total Number of Animals Assessed in Twenty-five Cities and Towns where the Disease was *Least* Prevalent, i. e., in the larger Cities and Towns.		Total Number of Animals Assessed in the Twenty-five Cities and Towns where the Disease was *Most* Prevalent, i. e. in the smaller Cities and Towns.	
Total population	943,614	Total population	142,639
Total number of cows	11,160	Total number of cows.....	12,801
1 cow to 84.5 inhabitants.		1 cow to 11 inhabitants.	
Total number of horses	29,519	Total number of horses....	10,352
1 horse to 32 inhabitants.		1 horse to 14 inhabitants.	
Total number of swine	3,288	Total number of swine.....	2,374
1 swine to 287 inhabitants.		1 swine to 60.5 inhabitants.	
Total number of fowls.....	41,887	Total number of fowls.....	79,797
1 fowl to 22.5 inhabitants.		1 fowl to 1.75 inhabitants.	
Total number of dogs.....	23,854	Total number of dogs.....	5,865
1 dog to 39.5 inhabitants.		1 dog to 24.3 inhabitants.	

Of course, this is only one of many conditions which are different in the two classes but the persistence year after year of this greater preva-

lence of the disease in the smaller cities and towns must mean something with regard to the etiology of the disease if we were only able to interpret it.

Seasonal Occurrence.—The occurrence of the disease by months shows that the maximum incidence of infantile paralysis occurs between June and November. In 1907 the largest number of cases occurred in September. In 1908 July and August had equal numbers; 1909 had the largest number in August, and 1910 in July. There seems thus to be in Massachusetts a distinct tendency of the maximum incidence to recede in point of time, a phenomenon which should it occur in 1911, might be worthy of serious investigation (see Charts 5, 6, 7, 8 and 9).

Age.—The combined experience of three years, 1908 to 1910, shows that 6.13 per cent. of the cases occurred before the end of the first year; 66.94 per cent. before the end of the fifth year, and 83.1 per cent. before the end of the tenth year. Moreover, the Massachusetts figures give the impression that the disease is more frequent in adults than has been commonly supposed (Table 6).

TABLE 6.—OCCURRENCE BY AGE PERIODS (1908-1910)

Age, Years	Cases	Per cent.
Birth to 12 mos., inc.	55	6.13
1	119	13.26
2	155	17.35
3	125	13.93
4	92	10.25
5	54	6.02
	600	65.94
6 to 10, inc.	145	16.16
	745	83.10
11 to 20, inc.	83	9.25
	828	92.3
21 to 30, inc.	39	4.34
31 to 72, inc.	17	1.89
Not stated	13	.....
	897	.....

Sex.—As far as sex is concerned, the relative incidence is expressed by the figures of 56 per cent. for males and 44 per cent. for females, as reported for the four years, 1907 to 1910 (Table 7).

TABLE 7.—SEX (1907-1910)

		Per cent.
Males	906	56.66
Females	689	43.08
Not stated	4	0.25
Total	1,599	.....

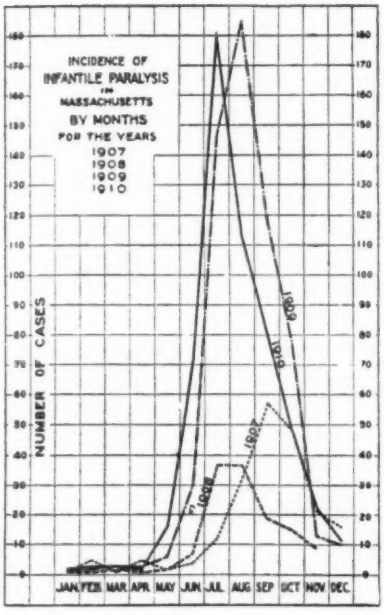

Chart 5.

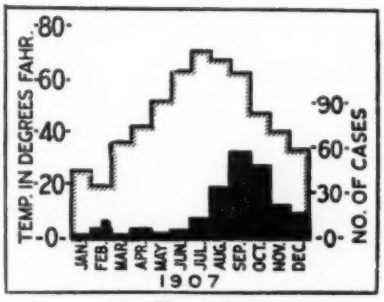

Chart 6

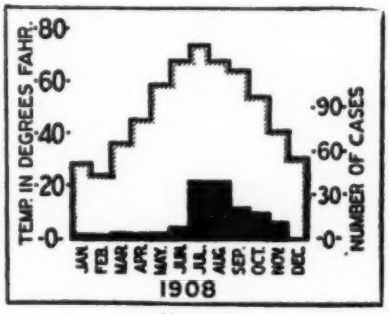

Chart 7

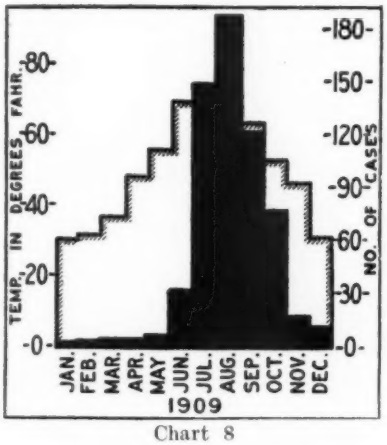

Chart 8

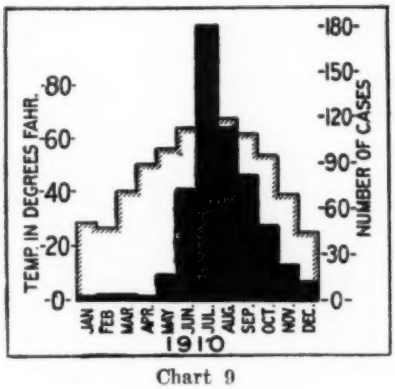

Chart 9

Chart 6.—Incidence of cases in 1907 by months shown in solid black; mean temperature in white.

Chart 7.—Incidence of cases in 1908 by months in solid black; mean temperature in white.

Chart 8.—Incidence of cases in 1909 by months in solid black; mean temperature in white.

Chart 9.—Incidence of cases in 1910 in solid black; mean temperature in white.

Mortality.—The mortality under 1 year is high, being for 1909-10 12.19 per cent. Between 1 and 10 years the fatality seems to drop, being 6.24 per cent., but after ten years the mortality again rises and reaches 14.81 per cent. The average mortality is 7.9 per cent. (Table 8).

TABLE 8.—MORTALITY BY AGE (1909-1910)

Age, Years	Cases	Deaths	Mortality, Per cent.
Under 1	82	10	12.19
1 to 10	945	59	6.24
Over 10	189	28	14.81
Totals	1.216	97	.....
Average mortality			7.9

Environment of Patients.—As regards the character of the house, practically an equal number of cases occurred in detached houses, in tenements or in apartments. Furthermore, there seemed to be no marked predilection of the disease for any special floor of the house. The house was said to be dry in 72 per cent., damp in 27 per cent., and medium in 1 per cent. No apparent relation to moisture can, therefore, be made out. The sanitary conditions were either excellent, good or fair in 85.6 per cent. cases, and bad in only 11.5 per cent. The age of the house was not remarkable, 41.14 per cent. of patients living in new houses; that is to say, within the first and tenth year of its existence; whereas 58.85 per cent. lived in houses more than ten years old.

It has seemed at times as if the disease preferred river beds, but this factor in the occurrence of the disease must rest on other basis than mere propinquity, because, although thirty-three patients are said to have lived near a river or near water, 317 lived more than one-eighth of a mile from any stream, pond, or beach, so that it seems probable that any marked occurrence of the disease along the water courses is due to other factors than a neighborhood of water, the determining influence being in all probability increased personal contact due to the fact that all highroads, electric railways and railroads are very apt to follow river valleys, and cities and towns are, therefore, more likely also to be situated along such water courses.

In the same way, no especial relation could be found between the occurrence of infantile paralysis and nearness to railroads. The experience of Wickman, as shown by his maps, certainly seemed to show a distinct preference of the disease for localities near railroads, but in Massachusetts we have not been able to observe any etiologic connection between the two. In 1909-10, 30.56 per cent. of the cases occurred within one-eighth of a mile of the tracks, while 69.42 per cent. were more than one-fourth of a mile away.

A study of sewage condition shows that the disease occurs under all conditions of sewage disposal and as regards water supplies from metropolitan and city systems, as well as from wells and springs (Tables 9, 10, 11, 12, 13, 14, 15, 16 and 17).

TABLE 9.—CHARACTER OF HOUSE (1907-1909)

		Per cent.
Detached	220	49.54
Tenements	224	50.45
Total	444	.....

TABLE 10.—FLOOR OF HOUSE INHABITED BY FAMILY (1907 AND 1909)

		Per cent.
The whole house	75	21.06
The first floor	139	39.04
First floor and basement	3	.84
Second floor	101	28.37
Third floor	32	8.98
Upper stories	6	1.68
Total	356	.....

TABLE 11.—LOCATION OF HOUSE (1907-1910)

		Per cent.
Dry	459	71.38
Damp	177	27.52
Medium	7	1.08
Total	643	.....

TABLE 12.—SANITARY CONDITION (1907-1910)

		Per cent.
Excellent	123	18.83
Good	271	41.50
Fair	165	25.25
Bad	74	11.33
Not stated	20	3.06
Total	653	.....

TABLE 13.—AGE OF HOUSE (1909-1910)

		Per cent.
New House (1-10 yrs.)	144	41.14
Old House (over 10 yrs.)	206	58.85
Total	350	.....

TABLE 14.—NEARNESS OF HOUSE TO WATER (STREAM, POND OR BEACH) (1909-1910)

		Per cent.
On river	33	9.42
Within ⅛ mile	66	18.85
Within ¼ mile	89	25.42
Within ½ mile	78	22.28
Within 1 mile	84	24.00
Total	350	.....

TABLE 15.—NEARNESS OF HOUSE TO RAILROAD (1909-1910)

		Per cent.
On railroad	57	16.28
Within ⅛ mile	50	14.28
Within ¼ mile	44	12.57
Within ½ mile	114	32.57
Within 1 mile	85	24.28
Total	350	.....

TABLE 16.—CHARACTER OF SEWAGE DISPOSAL (1908-1910)

		Per cent.
Sewage (Met.)	110	26.25
Sewage (City, etc.)	211	50.35
Cesspool	23	5.48
Vault	60	14.31
Privy	15	3.57
Total	419	.....

TABLE 17.—CHARACTER OF WATER-SUPPLY (1908-1910)

		Per cent.
Metropolitan	120	27.98
Town	62	14.45
City	172	40.09
Well	17	3.96
Spring	58	13.51
Total	429	.....

Rainfall.—The deficiency in rainfall during the last eight years has been, of course, very marked, and this factor may be of importance in the spread of the disease. It certainly has to be taken very seriously into consideration if we accept the suggestion of Hill of Minnesota that dust, especially dust infected with horse manure, may be responsible for the transference of this disease. The deficiency of rainfall in Massachusetts has varied since 1904, when it was 1.35 inches, to 9.42 inches in 1910. With the deficiency of rainfall during each year of this period has occurred a sort of cumulative effect, which makes this deficiency relatively more important each year. A study of the deficiency of rainfall shows that the largest number of cases have not occurred in the driest years in Massachusetts (Table 18 and Charts 10, 11, 12, 13).

TABLE 18.—DEFICIENCY RAINFALL (1904-1910)

Year	Cases in State	Actual	Normal	Deficiency
1904	...	43.81	45.16	— 1.35
1905	...	37.60	.....	— 7.56
1906	...	43.21	.....	— 1.95
1907	234	44.49	.....	— 0.67
1908	136	37.61	.....	— 7.55
1909	923	42.10	.....	— 3.06
1910	845	35.50	.....	— 9.42
				—31.56

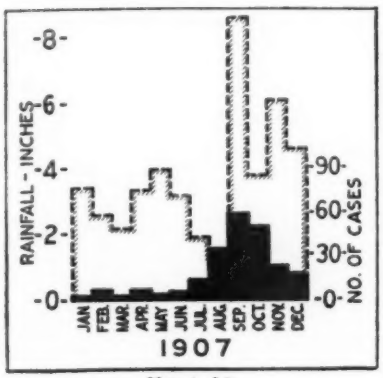

Chart 10

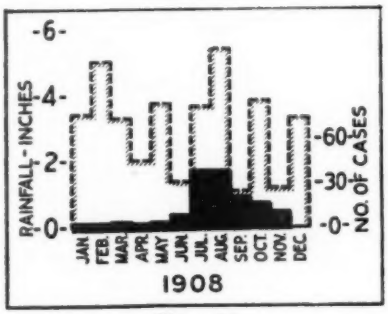

Chart 11

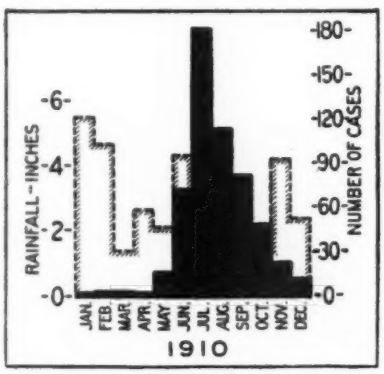

Chart 13

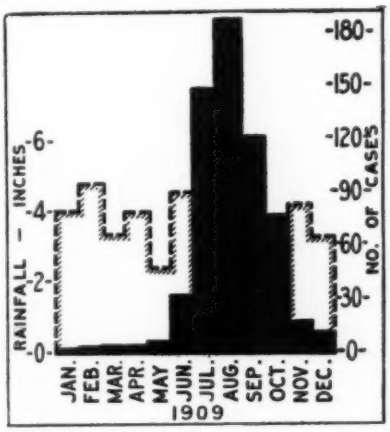

Chart 12

Chart 14

Chart 10.—Incidence of cases and rainfall by months in 1907. The former shown in solid black.

Chart 11.—Incidence of cases and rainfall in 1908. The former shown in solid black.

Chart 12.—Monthly incidence of cases and rainfall in 1909. The former in solid black.

Chart 13.—Monthly incidence of cases and rainfall in 1910. The former in solid black.

Chart 14.—Map showing endemic focus of infantile paralysis in a city of 119,000 inhabitants; cases occurring in the years 1903, 1908, 1909 and 1910.

Dust.—A study of the amount of dust in affected localities shows a report of a moderate amount in more than half of the cases while in 36 per cent. of the total number much dust or an excessive amount was reported. The matter is not, however, one capable of measurement or exact determination and the table cannot be regarded as important (Table 19).

TABLE 19.—RELATION TO DUST (1909-1910)

		Per cent.
No dust	0	.....
Very little dust	29	8.28
Moderate amount of dust	195	55.71
Much dust	118	33.71
Excessive amount of dust	8	2.28
Total	350	.....

Insects, Etc.—The possible transfer of the disease by vermin, insects, rodents, etc., was investigated, but the figures are not at all conclusive, and very little knowledge of value is likely to come in this department of the work, until individual infected localities are investigated by persons expert in the study of entomology, a line of investigation of the greatest importance (Table 20).

TABLE 20.—PRESENCE OF VERMIN, INSECTS, RODENTS, ETC. (1909-1910)*

		Per cent.
Flies	298	90.85
Mosquitoes	142	43.29
Mice (house)	123	37.5
Ants	97	29.57
Rats	77	23.47
Bedbugs	70	21.34
Roaches	64	19.51
Spiders	54	16.46
Mice (field)	24	7.31
Biting flies	10	3.04
Fleas	4	1.21
Grubs and Caterpillar	3	.91
Bees	2	.609
Sand fleas	1	.304
Snakes	1	.304
Crickets	1	.304
Squirrel	1	.304
Leeches	1	.304
Brown tail moths	1	.304
Moles	1	.304
Total	328 families	

*Report 1907 and 1908 only for flies and mosquitoes.

Domestic Animals.—The rôle played by domestic animals in the transfer of the disease is one of great possible interest, but as yet the interrelation of the paralyses of man with those of animals has not been sufficiently marked to justify any definite conclusion. Many cases of paralysis

in animals have been reported, but the instances in which paralysis in human beings has been coincident with such animal paralysis have been rare (Table 21).

TABLE 21.—DATA AS TO DOMESTIC ANIMALS (1909-1910)

		Per cent.
No animals of any kind in	131	39.93
Animals in	197	60.06
125 animals had sickness, paralysis or death in	70	21.34
Total	328 families	

Transmissibility.—The degree of contagiousness of infantile paralysis is a question of great theoretic and practical importance. It is one which at the present time it is practically impossible to analyze exactly because, associated with the paralytic cases of this disease, occur an unknown number of so-called abortive cases which may be of the greatest importance in transferring to others paralytic types of the affection. Furthermore, these unknown abortive cases become immune to subsequent infection, and if included in any study of comparative contagiousness, will go far, undoubtedly, to vitiate any conclusions drawn. Our figures show that in 350 cases carefully studied, there was direct contact with an acute case in 13 per cent.; direct contact with possible abortive cases in 1.42 per cent.; with chronic cases in 4.28 per cent.; and that there was, undoubtedly, indirect contact with an acute case through a third person in 4 per cent. of the cases (Tables 22 and 23. See Chart 14 for an endemic focus in a city of 119,000 inhabitants).

TABLE 22.—INSTANCES OF CONTAGIOUSNESS (1909-1910)

Certain direct contact:		Per cent.
With acute case	46	13.14
With possible abortive case	5	1.42
With chronic case	15	4.28
With both acute and chronic cases	2	.57
Certain indirect contact with acute case by third person	14	4.00
Number of cases investigated	350	.....

TABLE 23.—FAMILIES WITH MORE THAN ONE CASE (REASON FOR SUSPECTING CONTAGION) 1908-1910

		Per cent.
One case	357	92.24
Two cases	27	6.97
Three cases	3	.77
Total	387	.....

Institution Cases.—Forty-four institutions for children in Massachusetts were written to in 1910, in order to ascertain whether or not any cases of acute epidemic poliomyelitis had originated within those institutions during the year. One institution, containing 360 children, in

Fall River, reported a single case, a boy, 4 years old. No secondary cases occurred in this institution (Table 24).

Table 24.—Institution Cases	Cases	Incidence per 1,000
44 institutions with 3,600 children under 15 years of age	1	.277
Mass., with a population of 851,092 children under 15 years (Census of 1905) showed in 1910 cases of acute epidemic poliomyelitis under 15 years	532	.625

Children in institutions therefore would seem to be, in some measure and in some way as yet unexplained, protected from this disease. Their opportunities for contact with the outside world are obviously much curtailed in comparison with those of the ordinary child, and therein lies very probably the reason for their immunity.

Swimming, Etc.—Just prior to the disease, there was a history of swimming, wading, or paddling, in water more or less contaminated, in 25 per cent. of the cases just before attack (Table 25).

Table 25.—Swimming, Wading or Paddling (1908-1910)*

		Per cent.
Swimming, wading or paddling	105	25.05
No such history	314	74.94
Total	419	.....

*Water contaminated more or less in every instance.

Accidents.—In the same way, 34 per cent. of 457 cases gave a history of accidents, over-exertion or falls. With regard to the swimming, etc., the percentage would seem no higher than one would expect in any group of children at this season, living near the water. But the percentage of thirty-four accidents, falls and over-exertion just before the attack would seem perhaps unduly high (Table 26).

Table 26.—Accident, Over-Exertion or Fall Preceding Attack (1907-1910)

		Per cent.
Accident	41	8.97
Over-exertion	47	10.28
Fall	67	14.66
No such history	302	66.08
Total	457	.....

Other Diseases.—A study of the disease prevalent in affected towns coincident with infantile paralysis showed nothing of importance. A further study of recent antecedent illnesses in the patient showed merely a number of minor ailments of no especial significance (Table 27).

TABLE 27.—DISEASES PREVALENT IN TOWN AT TIME OF OCCURRENCE OF INFANTILE PARALYSIS (1909-1910)

	Cases	Per cent.
Not known	159	46.49
Infantile paralysis	120	35.08
Whooping cough	12	3.508
Measles	11	3.21
Gastro-enteritis	8	2.33
Diarrhea	6	1.75
Influenza	6	1.75
Tonsillitis	4	1.16
"Rheumatic fever"	4	1.16
Scarlet fever	3	.87
Mumps	2	.58
Malaria	2	.58
Pneumonia	1	.29
Coryza	1	.29
Chicken pox	1	.29
Typhoid	1	.29
Diphtheria	1	.29
Total	342	.....

Diet. — The relation of diet to infantile paralysis is possibly of importance, but as yet no definite relation between the disease and certain kinds of food has ever been made out. Although evidently unusual, the disease does occur in breast-fed babies, four cases of the kind having been recorded in our returns of the last two years (Table 28).

TABLE 28.—DIET (1909-1910)

		Per cent.
General	283	80.85
(a) Raw cow's milk	308	88.00
(b) Condensed milk	15	4.28
(c) Breast milk and other food	19	5.42
(d) Breast milk alone	4	1.14
Fish	265	75.71
Fruit	288	82.28
Berries	197	56.28
Meat	275	78.57
Canned goods; (a) fruits; (b) vegetables; (c) fish	202	57.71
Cereals	140	40.00
Bread and butter	23	6.57
Vegetables	275	78.57
Stews and soups	11	3.14
Eggs	186	53.14
Tea, coffee, cocoa	6	1.71
Malted milk	2	.57
Ice cream	83	23.71
Predigested and beef juices	4	1.14
Candy	27	7.71
Bananas	6	1.71
For cases numbering	350	.....

Symptoms.—Digestive disturbances are common before, during and after the attack. Diarrhea is less frequent, comparatively, than constipation (Table 29).

TABLE 29.—DETAILS OF DIGESTIVE DISTURBANCE (1909-1910)

		Per cent.
PRECEDING ATTACK		
Nausea and vomiting	120	34.28
Constipation	109	31.14
Colic	5	1.42
Diarrhea	39	11.14
Indigestion or stomach upset	4	1.14
Mucus in stools	2	.57
ACCOMPANYING ATTACK		
Nausea and vomiting	140	40.00
Constipation	149	42.57
Colic	15	4.28
Diarrhea	56	16.00
Indigestion (indefinite)	3	.85
Mucus in stools	1	.28
FOLLOWING ATTACK		
Nausea and vomiting	5	1.42
Constipation	122	34.85
Colic	2	.57
Diarrhea	18	5.14
Mucus in stools	2	.57
Data for cases numbering	350	.....

The analysis of the general symptoms of the acute attack in 720 cases show fever present in 88 per cent., brain symptoms in 37 per cent., and retraction of the head in 34 per cent. Sore throat was only recorded as present in 9 per cent. (Table 30).

TABLE 30.—GENERAL FEATURES OF ACUTE ATTACK (1907-1910)

		Per cent.
Fever	636	88.33
Pain and tenderness	528	73.33
Brain symptoms	266	36.94
Retraction	248	34.44
Sore throat	65	9.02
For cases numbering	720	.....

Appearance of Paralysis.—The appearance of paralysis in relation to the attack was studied in 589 cases. In 16 per cent. paralysis occurred on the day of the attack; in 16 per cent., one day after the attack; in 17 per cent., two days after the attack and in 16 per cent., three days after the attack. That is to say, in 65 per cent., within three days after the attack. In two cases it was recorded as coming before the attack and in twenty-three cases (4 per cent.) between one and two weeks after the onset (Table 31).

Pain and Tenderness.—The great importance of pain and tenderness as a symptom is not sufficiently recognized; 1,198 cases were analyzed in this regard and in 70.5 per cent. these symptoms were present. The disappearance of pain and tenderness in an analysis of 604 cases, in which the time was recorded, occurred in 30 per cent. within one week.

in 15 per cent. in from one to two weeks, and in 13 per cent. in from two to four weeks. In five cases it was recorded as lasting two to three months and in three cases as lasting several months (Tables 32 and 33).

TABLE 31.—APPEARANCE OF PARALYSIS IN DAYS AND WEEKS AFTER ONSET OF FEVER (1907-1910)

		Per cent.
Preceding attack	2	.33
Same day	95	16.12
One day	93	15.78
Two days	103	17.49
Three days	98	16.63
Four days	58	9.84
Five days	22	3.73
Six days	51	8.65
Seven days	18	3.05
Eight days	6	1.01
Nine days	2	.33
Ten days	4	.67
Eleven days	3	.509
Twelve days	5	.84
Thirteen days	1	.169
Fourteen days	4	.67
Not known (fatal)	1	.169
2 to 3 weeks	6	1.01
3 to 4 weeks	1	.169
4 to 5 weeks	1	.169
8 weeks	1	.169
Two days previous	1	.169
Not stated	13	2.207
Total	589	.....

TABLE 32.—PAIN AND TENDERNESS (1907-1910)

		Per cent.
No pain	178	14.85
Pain	845	70.53
Not stated	175	14.6
Total	1,198	.....

TABLE 33.—DISAPPEARANCE OF PAIN AND TENDERNESS (1909-1910)

		Per cent.
1 day or less	11	1.82
2 days	22	3.64
3 days	29	4.801
4 days	15	2.48
5 days	14	2.31
6 days	3	.496
A few days	28	4.63
1 week	59	9.76
1-2 weeks	91	15.06
2-3 weeks	46	7.61
3-4 weeks	33	5.46
4-5 weeks	1	.16
6-7 weeks	1	.16
1-2 months	28	4.63
2-3 months	5	.82
Several months	3	.496
Until death	39	6.45
Present when report was made.	175	29.13
Total	604	.....

Distribution of Paralysis.—The distribution of paralysis was studied in 1,158 cases. The most common location was in one leg, occurring in 28 per cent.; both legs came next in frequency with 24 per cent.; one or both arms in 9 per cent.; and where one arm and leg were affected, a hemiplegic distribution was more common than a crossed paralysis, the relative percentages being 10 per cent. and 3 per cent. Special attention should be called to the frequent occurrence of paralysis of the back, abdomen, neck and face, which are much more frequent than is supposed and too often overlooked (Table 34).

TABLE 34.—DISTRIBUTION OF PARALYSIS (1907-1910)

		Per cent.
One leg only	324	27.97
Both legs only	272	23.48
Back	154	13.29
Both arms and both legs	129	11.13
One arm and leg, same side	110	9.49
One arm only	84	7.25
Both legs and one arm	75	6.47
Face	74	6.38
Abdomen	67	5.78
One arm and leg, opposite sides	33	2.84
Respiration	31	2.67
Both arms only	23	1.98
Neck	11	.94
Both arms and one leg	10	.86
Deglutition	7	.604
Neck and back	6	.51
Ataxia (transitory)	5	.43
General	3	.25
Intercostal	1	.086
Both arms, back, chest and throat	1	.086
For cases numbering	1,158	.....

Prognosis.—In order to throw additional light on the question of prognosis, Dr. B. E. Wood of Boston was requested by the Board to investigate the present condition of all patients reported to the Board as paralyzed in the year 1907. This inquiry was made with especial reference to the number of recoveries occurring in the four years which had elapsed. Letters were sent to all physicians who had reported cases in 1907, asking whether or not such patients had recovered, and when recovery was reported, such cases were seen unless they had disappeared or moved, and if such was the case all available information was obtained about the patients.

Of 234 cases, twenty-two could not be investigated, eleven had died in the acute stage of the disease; eight others had died subsequently of other diseases. Although two of them are said to have recovered from the paralysis, no trace could be found of three cases. In 212 cases, therefore, the termination is known, and fifty-ceven of these (27 per cent.) are reported as having recovered.

On investigation it was found that there were three classes of recovery: (1) Complete recovery without atrophy, (2) recovery with complete function but with some atrophy, (3) recovery with some hypertrophy of the affected limb. The table shows the proportion of each of these.

TABLE 35.—CONDITION OF PATIENTS AFTER RECOVERY

		Per cent.
Complete recovery without atrophy	16	28.1
Functional recovery with atrophy	21	36.8
Recovery with some hypertrophy	3	5.3
Recovery, presence or absence of atrophy unknown	17	29.8

When atrophy was present, the maximum amount noted was as follows:

TABLE 36.—REMAINING ATROPHY AFTER RECOVERY

Calf	1¾ inches
Thigh	1¼ inches
Arm	½ inch
Forearm	¾ inch

In most cases the atrophy was much less than this, a difference of one-eighth inch being counted as atrophy, but in all such cases function of every muscle was perfect. The amounts of hypertrophy recorded were in two instances one-fourth inch and in one, three-eighths inch, all of the calf.

In these cases the severity of the attack was classed as:

TABLE 37.—SEVERITY OF THE ATTACK

		Per cent.
Severe	14	24.5
Moderate	9	15.9
Mild	28	49.1
Not noted	6	....
	57	....

The distribution of the paralysis in the fifty-seven recovered cases was as follows:

TABLE 38.—DISTRIBUTION OF THE PARALYSIS

One lower extremity	12
Both lower extremities	9
One upper extremity	6
Both upper extremities	2
One lower and one upper extremity	10
Three extremities	7
Four extremities	5
Face alone	1
General	1
Not given	4

In addition to this distribution, paralysis of the face also existed in four, of the back in five, of the face and back in one, and of the abdominal muscles in one.

The time of recovery in the fifty-seven cases was as follows:

TABLE 39.—TIME OF RECOVERY

1 week or less	2
1 week to 1 month	8
1-2 months	8
2-3 months	5
3-6 months	10
6-12 months	9
1-2 years	5
2-3 years	5
No data	5

CONCLUSIONS AS TO PROGNOSIS

The following conclusions seem justified as regards prognosis:

1. Complete recovery or functional recovery occurs in something over 25 per cent. of patients examined at the end of four years.

2. Atrophy may exist without impairment of function.

3. In about half of the recovered patients the onset was mild.

4. The distribution of the paralysis in such cases was not essentially different from that of cases which do not recover.

5. The period of recovery in many instances occupied months, and in several cases from one to two years.

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PYLORIC STENOSIS IN OLDER CHILDREN, WITH REPORT OF A CASE OF PYLORIC SPASM ENDING FATALLY

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It is not my intention to study in this paper the condition spoken of as congenital pyloric stenosis in infants, but to consider stenosis of the pylorus in children who have passed the period of infancy. The study of the condition in older children must, however, embrace those patients in whom the condition has persisted from infancy into later childhood, as well as those cases in which infancy has been free from all symptoms of pyloric stenosis, but in which the symptoms appeared months or years later.

At the outset of this study one is immediately impressed by the fact that while medical literature is crowded with articles on congenital hypertrophic pyloric stenosis and pyloric spasm in infants, there has been, as far as I am aware, little written exclusively on the condition in older children.

The discussion in all of these papers deals with the problem as to whether the condition is congenital or not; as to the cause of the hypertrophy of the circular muscular fibers at the pylorus; as to whether in a given case the condition is one of pyloric spasm simply, or hypertrophic stenosis simply, or both spasm and hypertrophy, and as to the necessity for an operation.

The etiology of these congenital or early cases of pyloric stenosis has been considered as due, perhaps, to gastric hyperacidity, or at least to some disturbance of gastric secretion and consequent gastric indigestion. Other causes are hyperplasia at the pylorus, edema of the mucous membrane of the pylorus and pyloric spasm. The causes of spasm are not clear. It may be due to a toxemia, to gastric dilatation, to a change in the gastric secretion, or it may be nervous in origin. The classical symptoms are vomiting, constipation, progressive loss in weight, scanty urine, visible peristaltic waves, and possibly finding a tumor at the pylorus.

The treatment ordinarily advised is, feeding, stomach-washing, opium, poultices to epigastrium, saline enematas for the purpose of absorption of liquid, nutrient enemata, operation.

A study of the subject of organic pyloric stenosis as found in American, English, German and French literature has impressed forcibly on me the belief that a careful clinical examination of children and young

adults will result in bringing to light a fair proportion of cases of organic stenosis during this period of life. The condition was long overlooked in infants; and while not flattering to clinicians, it is, I believe, nevertheless true that the condition is now being overlooked in the older child and young adult.

Children and young adults[1] who present gastric symptoms that continue for months or years, and especially if vomiting and epigastric pain are present, may be suffering from organic pyloric stenosis, and if such patients have been treated for a long time medicinally without benefit, the probability of there being organic pyloric stenosis increases. Many such cases have finally come to operation, and a pyloric stenosis found by the surgeon at operation.

The reason why medical treatment failed in these patients is then clear. Permanent changes have taken place in the gastric secretory apparatus, the result of the long continuance of the hypertrophic stenosis, and a cure by medical treatment is impossible. An early recognition of the pyloric obstruction should in a fair, or probably in a large, proportion of these cases lead to their cure by medical rather than surgical treatment. This seems at least probable, if one is to reason by the results obtained in pyloric stenosis in infants and older children. A study of the cases in infants and older children treated medically, shows conclusively that the majority of those infants who suffer from a mild degree of hypertrophic pyloric stenosis are cured by the physician and do not come to operation. Heubner[2] reports nineteen cured in twenty-one infants; Bendix[3] thirty in thirty-two cases; Hutchison[4] thirteen in fourteen cases. Stark[5] eleven out of twelve cases; Bloch[6] six patients, all recovered.

I do not wish to be understood as implying that there are not patients who require operation. I believe that the position taken on the question of operation by Robert Hutchison,[4] "that operation is never in any circumstances justified in these cases," is an extreme one, but that most of the cases of slight and moderate pyloric stenosis can be cured by medical treatment is my firm belief.

It is an accepted fact that many patients live into advanced adult life, who at autopsy show congenital pyloric stenosis. Rudolph Mayer[7] reports thirty-one such cases. Cautley and Dent[8] report three cases of pyloric stenosis at the age of 6, 11 and 22 years, respectively, and believe they

1. Maylard: Brit. Med. Jour., July 11, 1908, p. 71.
2. Heubner: Therap. d. Gegenw., 1906, viii.
3. Bendix: Med. Klin., 1909, vi, 1813.
4. Hutchison, Robert: Clin. Jour., Sept. 9, 1908.
5. Stark: Zentralbl. f. Kinderh., 1909, xiv, 5.
6. Bloch: Jahrb. f. Kinderh., 1907, lxv, 317.
7. Mayer, Rudolph: Virchow's Arch. f. Path. Anat., 1885, cii, 413.
8. Cautley and Dent: Tr. Roy. Med. and Chir. Soc., 1902, lxxxvi.

were probably congenital. Mayo Robson reports one case, Lauderer[9] ten and William Russell[10] three cases.

There is probably no doubt that congenital pyloric stenosis of a mild grade may remain latent for months and probably for years, and cases of organic pyloric stenosis have been observed during the second and third years of life, where a distinct insufficiency of motility, together with hyperacidity existed. There is probably hardly any year between early infancy and well advanced old age that does not show cases of pyloric stenosis in which the patients have come to operation. Some of these patients have had gastric symptoms from childhood; in others the symptoms vary from a few months to a number of years. Rosenheim[12] reports a case in a child who had always enjoyed good health until an attack of measles at the age of 5 years, from which he made a good recovery. Three months later the child began to vomit and continued to vomit almost every day. When 6½ years of age he was operated on and found to be suffering from hypertrophic pyloric stenosis. The case ended in recovery. Osler reports a case of hypertrophic pyloric stenosis in a child that lived until the third year.

There is much room for thought in the study of the cases in which the hypertrophic stenosis is not sufficient in amount to produce dangerous symptoms in infancy. The hypertrophy gradually increases in amount, the stenosis becomes more marked, and in later years the child presents the symptoms directly and indirectly produced by the stenosed pylorus. Beardsley reports a case at the age of 4 years, Sonnenberg one at 5 years, and Hansy one at 11 years (Cautley[13]).

Then there are cases that present exactly the opposite picture. The symptoms of hypertrophic pyloric stenosis are very severe in infancy; the babies are so ill that it seems as if they must surely die. They recover, however, and at the age of 3 or 4 years are apparently perfectly well. Robert Hutchison has reported such cases.

The question naturally arises, what has occurred in these cases? Has the congenital hypertrophy of the pylorus largely or partly disappeared? Has the compensatory hypertrophy of the stomach muscles been sufficient to overcome the original condition, and has no gastric dilatation occurred, or has it been a combination of both conditions? Cases are reported in which apparently no gastric dilatation has occurred, and the evidence in such cases points strongly to absorption of the hypertrophy.

What is the explanation of the very large percentage of recoveries reported by certain observers — Heubner, Bendix, Hutchison, Stark, and

9. Lauderer: Tübingen, 1879.
10. Russell, William: Brit. Med. Jour., July 11, 1908.
11. Bernheim (Zurich): Deutch. med. Wchnschr., 1908, p. 1830.
12. Rosenheim: Berl. klin. Wchnschr., 1899, xxxii, 703.
13. Cautley: Diseases of Children, 1910, p. 264.

Bloch? I believe it is as follows: Many of these cases are instances of pyloric spasm; others are cases of slight hypertrophic pyloric stenosis; in still others, both spasm and a mild degree of hypertrophy exist, and if one is ready to admit that a slight amount of hypertrophic pyloric stenosis may be largely or in part absorbed, the explanation is quite satisfactory. Stiles, who has operated on many of these patients, claims that true congenital hypertrophic stenosis of the pylorus is rare, and when it does occur, should be treated surgically. Cautley is of about the same opinion, but believes that very mild cases of this disease may recover under medical treatment. Cases of this character may, and probably do, in some instances, show gastric symptoms and evidence of pyloric stenosis in later life, since persistent stenosis in the older child is represented by, first, a period of compensatory stomachic muscular hypertrophy; this is followed by a period which may exist from the inception of the stenosis which represents stagnation and the slow emptying of the stomachic contents; and lastly, retention, or the failure of the stomach ever to completely empty itself. Is it not possible to recognize these cases clinically? Should they come to operation undiagnosed?

The following conclusions in regard to hypertrophic pyloric stenosis in older children and young adults seem to me to be justifiable:

1. Pyloric stenosis is present in children and young adults more commonly than is supposed.

2. The age at which it manifests itself depends on the degree of stenosis present.

3. Pyloric stenosis may be latent for years.

4. It is found by the surgeon during childhood and young adult life, and its early recognition by the physician is important from the standpoint of early medical or, if necessary, surgical treatment.

5. The entire disappearance of all the classical symptoms of congenital hypertrophic stenosis, and the apparent health of the infant during its subsequent early childhood, suggest the probability of an absorption of the hypertrophy, especially as no gastric dilatation may develop later in life.

CASE REPORT

It may be interesting in this connection to give the history of a case of pyloric spasm in a boy aged 6½ years, whose general health during infancy and childhood had been first-class until a few months before his death; who presented during the last few months of his life a typical picture of pyloric spasm; who came to autopsy, and in whom no disease of the pylorus or stomach was found post mortem.

History.—W. L., aged 6½, was admitted to the Jefferson Hospital March 8, 1911, under the care of Dr. Nassau, and I was asked by Dr. Nassau to see him a few days after his admission. The history of the case was as follows: Mother, father, two sisters, and two brothers were in good health; no hereditary history.

The child had pertussis one year previously. The left eye was injured by a fall in July, 1909, and the eye was enucleated in November, 1910; he gained 9 pounds in three weeks after the operation. Six weeks previous to admission to Jefferson Hospital he began to vomit. Vomiting occurred at irregular periods, bore no relation to the taking of food, and was most frequent in the morning. Vomiting was preceded by nausea and restlessness, which was relieved by vomiting. Vomiting might be absent for several days. He had no pain, no blood in the vomitus; had been rapidly losing weight, and was becoming much weaker, and had been confined to bed for one week. There was no history of chills or fever.

Examination.—The patient was poorly nourished; pupil of right eye was large, but reacted well to light and accommodation; a few submaxillary glands were palpable on both sides; tongue coated, with clean, pink margins. Chest poorly developed; ribs prominent; lungs normal. Abdomen was scaphoid; walls thin; no areas of tenderness or rigidity; no pyloric tumor could be felt. Urine, acid, 1,010, no albumin, no sugar, no acetone or diacetic acid; urea 2.9 per cent.; few leukocytes; no blood, no casts. March 17, 1911, the child had vomited three times in the previous two days; no vomiting had occurred during the two days previous to that; there was constipation; weight with night clothes and sandals, 35¾ pounds. The mother stated that he weighed 57 pounds two and one-half months previously. Report on the vomitus showed no hyperacidity and that bile was present. March 20 a bismuth meal was given. An *x*-ray plate seven hours after the meal showed the bismuth distributed in the stomach, but no trace of bismuth in the duodenum. This plate proved conclusively that food failed to pass normally from the child's stomach in the usual period, as a normal stomach will clear itself of a bismuth meal in from five to seven hours. Another plate showed the gold ball which had been placed in the orbit after enucleation of the eye. I did not learn until afterward that the eye was removed for sarcoma. The stools were small, yellow and well digested. The amount usually vomited at one time was never much more than the amount taken at one feeding. The Morro test was negative.

March 21. The child vomited first Jan. 20, 1910. The vomiting gradually got worse; formerly there was less effort in vomiting, but now vomiting was more violent and retching. He had been constipated all his life. The patient took and retained 14 ounces of water; no tumor was palpable with the stomach so distended. Examination of the unoperated eye showed no lesion of the fundus. The pulse was 130 to 150; temperature 97 F. to 102 F. The treatment consisted of liquid nourishment, nutrient enemata, stimulants, bromid of soda and alkalies. As the patient was not improving under medical treatment, and there was evidently from the *x*-ray plates a growth developing in the eye that had been previously operated on, and especially in view of the fact that I had learned that the eye had been operated on for sarcoma, a second operation on the eye was performed by Dr. Howard Hansell March 25, 1911.

Operation on the Eye.—The orbital cavity was completely filled with a dense hard growth, irregular in outline and extending forward to the commissure. The overlying conjunctiva appeared to be healthy. In operating for the removal of the growth, a horizontal incision was made through the conjunctiva from one canthus to the other. Through this opening the finger was introduced and by manipulation the tumor was enucleated and removed in sections. The largest portion included in its center the shrunken sclera, which partly enclosed a gold ball which had been inserted at the time of the enucleation of the globe some months previously.

The floor of the orbit was extensively necrosed and communicated by a large rough-edged, round opening with the antrum of the same side. The floor and outer wall of the orbit were denuded of periosteum, and contained several necrotic areas. The sphenoidal fissure was much larger than normal, by reason of adjacent diseased bone.

The patient rallied fairly well from the operation, but continued to grow weaker; vomiting continued; the amount of nourishment taken was very small, and he died April 6.

Necropsy.—Post-mortem record of W. L. Date of post-mortem, April 7, 1911, 12 noon.

Pathologic Diagnosis.—Sarcoma of liver, enlarged mesenteric lymph-nodes.

External Examination.—Body is that of an extremely emaciated male child. The left orbit contains a small quantity of gauze, which is saturated with purulent material. Rigor mortis present but easily broken up. Post-mortem suggillation is marked posteriorly.

Internal Examination.—Musculature wasted and subcutaneous fat very scant.

Peritoneum: Smooth and glistening; cavity contains a small amount of clear, straw-colored fluid; liver extends to costal margin, mid-clavicular line. The intestine is partially collapsed; this condition to be described later.

Left Pleura: Free from adhesions; surfaces are smooth and glistening; cavity contains a small amount of straw-colored fluid.

Right Pleura: Resembles its fellow of opposite side.

Pericardium: Smooth and glistening; cavity contains small amount of clear serum.

Heart: Normal in size. Right auricle—endocardium smooth and glistening; cavity filled with semifluid blood.

Left Ventricle: Wall is 0.6 cm. thick; musculature red and firm; cavity filled with semifluid blood.

Left Lung: Crepitates throughout; cut surface pinkish-red in color, and shows no gross lesions; weight, 160 gm.

Right Lung: Same. Weight, 150 gm.

Spleen: Measures 6x3x2 cm., is soft and of purplish-red color; weight, 160 gm.

Left Kidney: Measures 6x3.5x2.5 cm.; red in color; cuts with normal resistance; cortex bears normal relationship to pyramids and is red and injected; capsule strips readily, revealing a smooth surface; weight, 75 gm.

Right Kidney: Resembles its fellow; weight, 70 gm.

Ureters and Bladder: Show no gross lesions.

Stomach: Contains a small amount of yellowish material; capacity of 175 c.c. The mucous membrane shows some slight post-mortem discoloration. The pyloric orifice is patulous, and the pyloric ring seems to be normal in thickness.

Duodeum: Mucosa bile-tinged, but shows no macroscopic lesions.

Liver: Normal in size, reddish color, capsule smooth, cuts with normal resistance. In the right lobe near the anterior margin there is a firm, yellowish nodule measuring 4 cm. in diameter, sharply demarcated from the surrounding liver tissue. Remainder of organ apparently normal.

Pancreas: Normal in size and color, and shows no noteworthy lesions.

Intestines: Mesenteric lymph-nodes are enlarged, firm and of reddish color. The small intestine at a distance of 20 cm. from the ileocecal junction is collapsed for a distance of about 50 cm. Remainder of organ shows no lesions.

Vena Cava and Aorta: Normal.

Microscopic Diagnosis.—Tumor of Liver: Small round-celled sarcoma.

Pylorus: Normal.

Mesenteric Glands: Hyperplasia of lymphoid tissue.

Brain: Showed nothing.

The case of pyloric spasm here reported and coming to autopsy is to me of great interest. It was unconnected with any organic disease of the stomach, or with any change in the normal gastric secretion. Spasm of the pylorus in older children is spoken of by many authors, and is usually described as being associated with severe pain, perhaps

little or no vomiting, and often connected with disease of the pancreas, appendix or gall-bladder, gastric ulcer, or distinct change in the gastric secretions. Pain was absent in my case; vomiting was the one prominent symptom, and loss in weight was rapid and extreme. In these two particulars, absence of pain and violent and persistent vomiting, it differs from the cases described by most writers. After the enucleation of the eye, the vomiting lessened, and in three weeks the boy gained 9 pounds. The vomiting ceased for five or six weeks after the first operation, and then gradually increased in frequency and severity until the second operation, when the sarcomatous mass was thoroughly removed from the orbit.

The case appears to belong to the group of so-called nervous vomiting, of reflex origin, associated primarily with disease of the eye, and later, aggravated by metastasis in the liver. Reflex vomiting of eye origin is independent of the taking of food, and while it lasts is persistent. The vomiting of eye origin does not last for days, and is usually followed by an ability to take and retain food. The primary operation was followed by entire cessation of vomiting, and a gain in weight. The sarcoma returned in the orbit, metastasis occurred in the liver and both these conditions caused, in my opinion, a severe return of this gastro-neurosis. The operation of cleaning out the orbit only slightly affected the vomiting; the boy was in a deplorably weak condition at the time of the operation, and the sarcoma of the liver was probably instrumental in causing a continuance of the vomiting, which persisted, although in a less degree, until his death. In my opinion, the second operation neither hastened or retarded death.

The stomach through the sympathetic and cerebrospinal nerves is connected with practically all organs and tissues in the human body, and it has been demonstrated that pyloric spasm can be produced artificially by stimulation of the vagus. The contraction of involuntary muscular tissue, the source of the contraction being outside the stomach, is at least probable.

Is pyloric spasm unassociated with pain and with persistent vomiting, a common condition, or even an occasional condition in older children? It certainly clinically does not resemble the cases described as associated with gastric ulcer,[14] and gall-stone colic in which the pain is excessive and vomiting not a marked symptom. Periodic pyloric spasm may occur at the menstrual period,[15] unassociated with any disease of the stomach, or with spasm in any other portion of the body, and under such conditions is probably a primary spasm of the pylorus, a motor neurosis. Lauder Brunton's observations tend to show that pyloric spasm may accompany migraine, and be a manifestation of a neurosis.

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14. Fairchilds: Iowa Med. Jour., Des Moines, 1910-11, xvii, 212.
15. Hemmeter: Diseases of the Stomach, Ed. 3, p. 744.

A number of cases of infantile pyloric spasm have been followed up to the age of 5 years and older, and found to be in first-class health, and a neurotic family history is not found in quite a considerable number of such cases. The clinician should not lose interest in these babies after they pass beyond the period of infancy, and an effort should be made to follow their subsequent histories, especially with reference to any gastric symptoms.

Cases of infantile pyloric spasm have been kept under observation for years.[17] All the symptoms may persist until the third year, and the child may remain nervous and anemic for years afterward. In other cases, solid food cannot be given until the child is 3 or even 5 years old. In still other cases of spasm, vomiting may persist until the child is 4 years old or older, and the peristaltic waves may continue until the child is over 4 years of age.

Cases diagnosed as cyclic vomiting have come to autopsy[18] and the lumen of the pylorus has been found to be very small, with a distinct hypertrophy. In these cases the children may be 4 or 5 years of age, the symptoms of epigastric pain and vomiting having been present since birth, returning at irregular intervals of months, and the children being apparently perfectly well between the attacks.

I do not mean to give the impression that cyclic vomiting and pyloric spasm are not two entirely different conditions, but to suggest the advisability of considering the possibility of pyloric spasm producing symptoms that resemble closely those of cyclic vomiting.

What are the causes of the pylorospasm in infancy that disappears in later childhood? Half of these children are breast-fed, and it seems to me unnatural to claim, as does von Starck, that the unaccustomed presence of food in the stomach produces an irritation of the gastric mucosa and so causes the spasm.

A clearer understanding of the etiology in infants may help us in the recognition and study of the condition in older children.

1713 Spruce Street.

16. Von Stark: Zentralbl. f. Kinderh., May 1, 1909.
17. Heubner: Berlin. Therap. d. Gegenw., 1906, viii, 433.
18. Russell: Proc. Roy. Soc. Med., London, 1909-10, iii, Sect. Dis. Child., 78.

AN APPARATUS FOR THE COLLECTION OF THE EXCRETA OF INFANTS*

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NEW YORK

The collection of the excreta of male infants is not a difficult matter. All that is necessary is to immobilize the child's pelvis, adjust the proper receptacles and let nature take its course.

Many kinds of apparatus have been used for this purpose and there is quite an extensive literature on the subject. This is well reviewed by Schabad[1] and by Langstein,[2] who also describes the technic of the investigation of the problems of the metabolism of children. Most of the devices

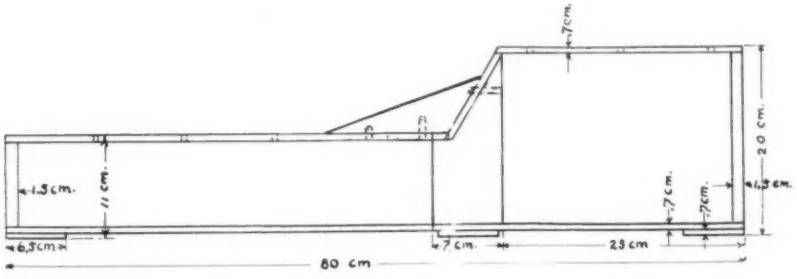

Fig. 1.—Diagram of apparatus for holding infants in metabolism experiments, showing measurements and method of construction.

for collecting urine go back to the girdle and test-tube principle of Raudnitz.[3] The devices for holding the child in one position have their origin in the hammock principle of Bendix,[4] modified later by Bendix and Finkelstein.[5] The hammock is tied to the ends and sides of the crib and the child fastened with his body resting on a support made of pillows, his legs spread apart and immobilized, his buttocks slung free over a dish or rubber cloth and his penis placed in a tube which leads to a glass

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*From the Pathological Laboratory of the Presbyterian Hospital and the Department of Applied Pharmacology, Cornell University Medical College.

*Demonstrated before the Section on Medicine New York Academy of Medicine, May 17, 1910.

1. Schabad, J. A.: Arch. f. Kinderh., 1908, xlviii, 402.
2. Langstein, Leo.: Handbuch der biochem. Arbeitsmethoden, 1910, iii, 1016.
3. Raudnitz: Prag. med. Wchnschr., 1883, viii, 359.
4. Bendix, B.: Jahrb. f. Kinderh., 1896, xliii, 23.
5. Bendix and Finkelstein: Deutsch. med. Wchnschr., 1900, xxvi, 672.

receptacle. An American modification by Talbot[6] makes use of a Bradford frame with a round hole for the buttocks.

A limited experience with several of these devices showed that it was necessary to use a good deal of restraint and a good deal of vigilance or the baby would get out of position. The child, although he was really warm and comfortable, looked chilly and uncomfortable. This roused unfavorable comment from nurses, parents and visitors. The apparatus of Howland and Cooke[7] is free from most of these disadvantages and does its work with absolute accuracy. It is easy to make and is perfectly

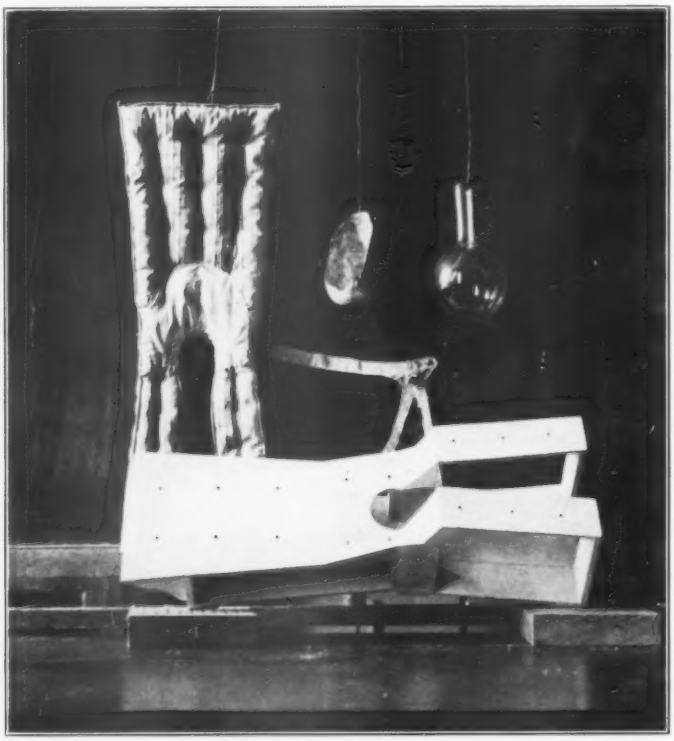

Fig. 2.—The complete apparatus, including mattress, receptacles for holding feces and urine and the girdle for holding the latter.

comfortable for the baby, but has the disadvantage of being attached to the crib. This makes it bulky and hard to move and also somewhat conspicuous to anxious parents. Still it is probably the most convenient form of apparatus for much of the work done with children.

From the necessities of my work I was forced to make an apparatus (Figs. 1, 2 and 3) which was portable and inconspicuous. It seemed

6. Talbot, F. B.: Jour. Am. Med. Assn., 1909, liii, 1818.
7. Howland, J., and Cooke, R. A.: Am. Jour. Dis. Child., December, 1911.

desirable also to keep the baby with thighs flexed, as this is the natural position for infants and is the position adopted in most of the recent devices. A frame of wood covered with a mattress was therefore modeled to fit the baby with thighs flexed. An inclined plane under the thighs prevented downward motion, a cloth band over the thighs prevented upward motion and a rounded trough in which the pelvis lay, prevented lateral motion. Beneath the buttocks was an opening with a receptacle for feces. Between the legs there was ample room for the neck of a flask to hold the urine.

The babies did well on this bed and seemed to enjoy the additional attention and the entire absence of wet diapers. The whole affair was so small that it could be placed in a bassinette and so flat that no one would

Fig. 3.—The apparatus assembled and the infant secured in place.

suspect the child was restrained in any form of apparatus. It could be easily wrapped in a blanket and carried outdoors or held in a mother's lap while the infant nursed. When the proper receptacles were used, there was absolutely no loss of urine or feces. I have never seen children kept more than three days on the apparatus, but there is no reason why the period should not be extended. There are very seldom any pressure signs.

The accompanying pictures show the construction of a frame which will hold children weighing up to 8 kilos (17½ pounds), but is somewhat large for very young babies. It is made of light wood 0.7 cm. in thickness, and is carved so as to fit the pelvis and buttocks. White enamel paint makes the best finish. The mattress should be about 5 cm. in thick-

ness and should be securely bound to the frame by strings passing through the holes bored in the top of the frame. It is particularly important to have the mattress form a good hollow for the buttocks. The edges of the hole under the buttocks should be protected by sewing a dress shield to the mattress. The best feces receptacle is a sugar scoop with the handle cut off, attached by a wire to a hook on the under surface of the frame. Instead of the sugar scoop a deep enamel-ware cup may be used.

The girdle for the urine receptacle is shown in the photograph. A thick-lipped test-tube which fits comfortably over the boy's penis is bent, drawn out and cut off at the constricted portion. This is passed through the hole in the girdle and secured to it by means of a wire. To protect the scrotum, the penis is passed through a hole in a square piece of non-absorbent cotton, is smeared with vaselin or boric ointment and placed in the test-tube. The girdle with its leg bands is then adjusted and the test-tube arranged so as to empty into the neck of a large flask. It is better not to use rubber tubing if it can be avoided. In the flask should be placed a crystal of thymol to prevent decomposition of the urine. The flask is easily suspended from the under surface of the frame by wires. The urine flows better if the head of the frame is raised a little.

To hold the child on the frame one should use broad cloth bands around the thighs and around the abdomen. This lets the baby kick his legs, wave his arms and wriggle his thorax and neck. It is wise to put a pillow on either side of the frame to help keep the child from wriggling too far.

The frame and mattress can be made at a cost of about eight dollars. Once made it lasts indefinitely and can be used for many babies. I have not tried to use it with female infants and have not yet tried it in surgical cases which demand restraint.

129 East Seventy-Sixth Street.

A METABOLISM BED FOR INFANTS*

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The following is a description of the metabolism bed that we have used during the past two years. It is very simple and comfortable and gives excellent results. For this reason it is described.

The bed proper is one in general use in New York. It is made of iron, with a movable spring mattress, which may be raised or lowered at the head or foot (Fig. 1). During experimental periods the foot of the mattress is lowered at a decided angle as shown in the picture. A stout frame, made of half-inch gas-pipe, 16 inches wide and 3½ feet long, is allowed to rest on the mattress at the head of the bed and is fastened firmly to the bars so that it will not slip toward the foot. Below, the frame is suspended by straps or tape to the sides of the crib so that the foot is slightly lower than the head. This inclination is of advantage in making the collection of urine and feces easier, and the slight tendency of the child to slip toward the foot is of assistance in holding him in a proper position. The greater angle at which the mattress is inclined leaves a space between the mattress and the frame in which the receptacles for the collection of urine and feces may readily be placed.

The upper part of the frame is covered by a piece of stout canvas about 16 inches wide, drawn very tightly and smoothly and laced on the under side of the frame.

A special retaining sheet is cut as shown in the diagram (Fig. 2). Its size must depend somewhat on the size of the infant used for study. In general the hole for the buttocks should be 5 inches in diameter and those for the legs 3 inches. The upper part of the sheet is torn to form strips, like the ordinary scultetus binder. The last strip should come a little below the level of the hole for the buttocks, as this holds the buttocks more firmly in place. The holes in the sheet are faced with canvas on the under side and strips of canvas are continued down the sheet to a point a little below the level of the leg holes in order to strengthen the sheet at the places of maximum strain. The sheet is roughly stitched to the canvas covering of the frame, care being taken to have the hole for the buttocks about 2 inches below the lower end of the canvas. A wooden bar is fastened across the bed to the side of the crib just above the lower end of the canvas.

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*From the First Medical Division of Bellevue Hospital, Children's Service.

The child is prepared as follows: A woolen jacket protects the body. Cotton is bound about the knees to protect them from pressure by the leg holes, and in cold weather the legs and feet may be completely covered by cotton held in place by gauze, with stockings drawn over all, or the infant may be dressed in a woolen night dress with legs.

Male babies only can be used. The penis is placed in a glass or metal tube, held against the symphysis by a rubber waistband with perineal straps and connected with the collecting flask below by rubber tubing. Glass tubing has proved most satisfactory for the penis as it can easily be varied in size and shape, is readily cleaned and allows one to see that the position is proper.

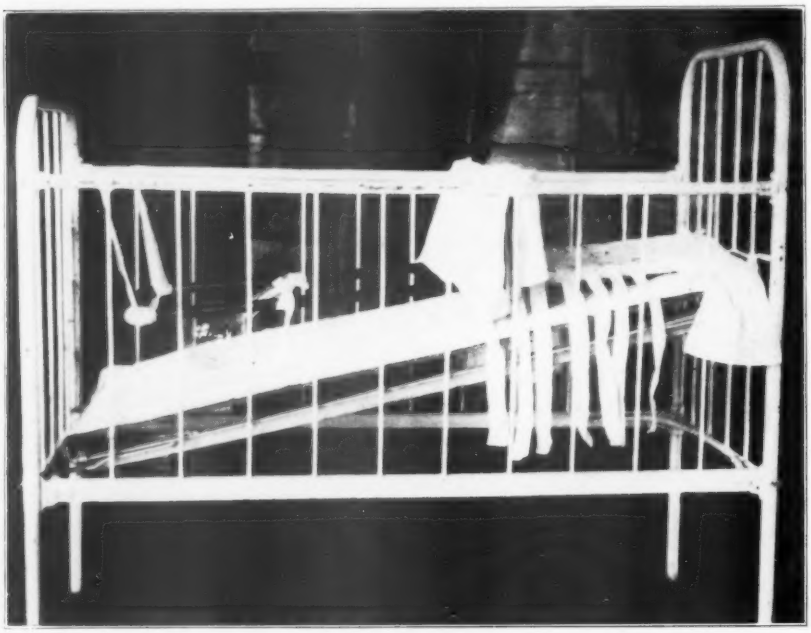

Fig. 1.—Metabolism bed, showing position of spring mattress frame, collecting flask, etc.

The child is then placed on the frame, the legs drawn through the leg holes to the knees, the buttocks well down in their proper hole, and the scultetus binder is loosely tied. The lower part of the sheet is then drawn up to the cross bar and pinned or tied as shown in the diagram. The feet are supported by a hammock of gauze swung across the bed parallel to the cross bar and a little below it. A large porcelain dish is put under the buttocks and the tube from the penis is placed in the glass flask fastened in the frame as shown in the picture. Pillows or blankets are then placed around the inside of the bed to protect from draught, and hot water bottles, if necessary, may be added.

A satisfactory metabolism bed must be simple, efficient and comfortable. The frame and sheet of the bed described above are easily made and it takes but five minutes to put a child on the frame. We can also vouch for the efficiency of this bed. But above everything, the comfort of the patient must be provided for, for only in this way is a normal, or rather, representative, metabolism maintained. It is particularly this feature which has commended this bed to us above the many others described. The child lies in a perfectly natural position with slightly flexed thighs and with absolute freedom of the arms and legs, and these

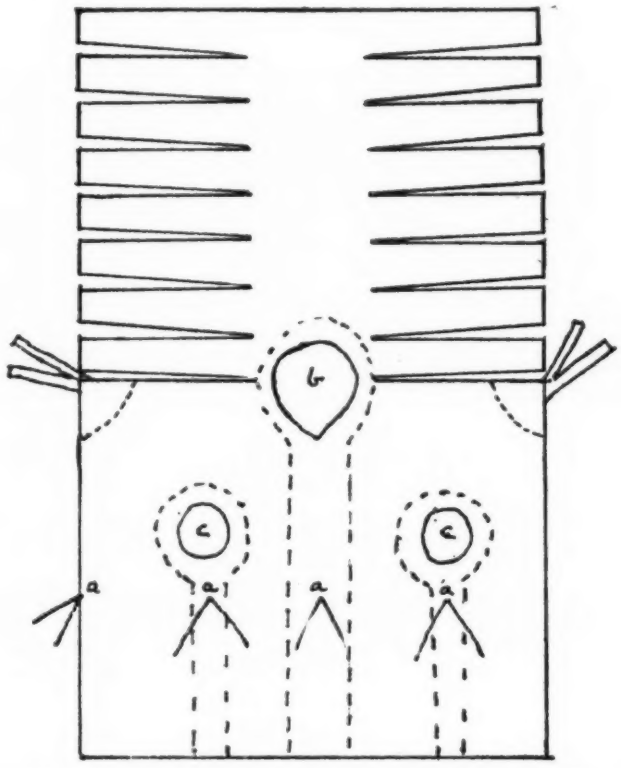

Fig. 2.—Diagram of bed sheet. Dotted lines show reinforcement with canvas; a, tape for tying to cross-bar; b, hole for buttocks; c, holes for legs.

are at all times supported so that he can move them at will or allow them to lie still without any strain on them. He can, moreover, turn somewhat from side to side, if the binder is loosely tied, without the risk of disturbing the collection of excreta.

We have kept infants for days on this frame without the slightest evidence of fatigue or resentment at restraint.

24 West Fiftieth Street.

PROGRESS IN PEDIATRICS

DISTURBANCES OF NUTRITION OF THE ARTIFICIALLY FED INFANT WITH PRACTICAL APPLICATION OF THE CLASSIFICATION OF FINKELSTEIN

JULIUS H. HESS, M.D.
CHICAGO

In order to make the subject more comprehensible, I take the liberty of reviewing the etiology of nutritional disturbances as classified by Langstein and Meyer.

ETIOLOGY IN GENERAL OF NUTRITIONAL DISTURBANCES

VARIETIES

1. Food qualitatively normal, but metabolism abnormal. Idiosyncrasy, as seen in (1) neuropathic infants; (2) exudative diathesis.

2. Milk infected with bacterial products due to the action on fats and sugars. (These products are not affected by sterilization.) Seen most in (a) summer, and (b) unhygienic milk.

3. Primary infection. Dysentery, typhoid, streptococcus enteritis, etc.

4. Secondary infection. Probably primary injury of the sensitive intestinal epithelium, a counterpart of the nephritis which is so frequently seen following colds, pharyngitis, otitis, pneumonia, pyelitis, cystitis, etc., in sick infants.

5. Inanition, as seen in (a) pyloric stenosis; (b) excessive starch feeding (*Mehl-nahrschaden*).

6. Overfeeding. Not necessarily based on an excessive fuel value of the ingested food, but frequently due to a preponderance of one or more food elements, either alone or in the presence of a vitiated digestive apparatus.

It is the latter problem, i. e., *overfeeding,* which we will discuss, referring to the previously named etiologic factors only in so far as they may directly have a bearing on the cases under discussion.

HISTORY

The history is of vast importance in all nutritional disturbances and worthy of the most careful consideration. The following should be carefully noted: the constitution of the child, with the presence of hereditary and developmental defects, a neuropathic history, or the presence of an exudative diathesis; lowered immunity, as evidenced by previous general, local, or gastro-intestinal infections; history of improper feeding; quality or quantity of individual feedings and total food taken, and the frequency and number of feedings, with notation of the resulting effect on weight and temperature curves. Briefly stated, in a normal child an increase of food results in increased weight, normal temperature, and normal

stools; while in nutritional disturbances increase of food results in decreased weight, increased temperature, and often abnormal stools, a paradoxical reaction.

The effects of the individual food elements in excess in nutritional disturbances are as follows: *Salts* cause a rise in temperature; *sugars* cause toxic symptoms, rise in temperature, and diarrhea; *fats* cause toxic symptoms, obstipation and usually fat stools (possibly diarrhea); *proteins* cause no corresponding findings. Results are not due to the individual ingredients, but to their relations to each other in a given food.

Based on the above etiologic factors and metabolic facts, Finkelstein has divided nutritional disturbances into the following types:

I. Disturbed metabolic balance (Bilanz Störung).

II. Dyspepsia.

III. Intoxication.

IV. Decomposition (acute atrophy).

Nutritional disturbances should not be considered as local intestinal lesions, but as general, deep-seated disturbances of the whole organism.

I. DISTURBED METABOLIC BALANCE (BILANZ-STÖRUNG — FINKELSTEIN; MILCH-NÄHRSCHADEN — CZERNY-KELLER; ATROPHY OF MODERATE DEGREE)

1. Exogenic Factors.— Improper nourishment (usually excess of fat in food). Under this head we will consider in detail the disturbances described by Czerny-Keller under the name of *Milch-Nährschaden* (nutritional disturbances due to excessive milk feeding), which usually arise during the course of feeding with ordinary milk mixtures, the clinical picture usually being due to the fat content, which may be relatively or absolutely in excess.

2. Endogenic Factor.—Congenital idiosyncrasy toward milk digestion. This is clinically characterized by

(1) *Weight:* Notwithstanding proper or even excessive caloric intake, there may be no gain in weight or an irregular increase, however, under the normal.

(2) *Temperature:* Varies as to the causative factor. Usually we find a daily oscillation from 1 to 2 degrees, with a tendency toward subnormal. However, in the presence of associated excessive carbohydrates there is an early hyperpyrexia due to the development of dyspepsia. This is especially true of infants under six months.

(3) *Stools:* In excessive milk feeding (*Milch-Nährschaden*) the common type is the fat soap stool, which is dry, light in color (gray to white), friable, and does not stick to the napkin. In the presence of excessive carbohydrates this stool may be lacking, due to the presence of a slight dyspepsia.

(4) Regurgitation and vomiting are frequent.

(5) Skin is pale, with loss of elasticity.

(6) Muscles are soft and flabby.

(7) Abdomen tympanitic.

(8) Child restless.

(9) Sleep disturbed.

(10) Immunity lessened, with resulting furunculosis, eczema and intertrigo.

(11) Urine usually shows no marked changes.

Metabolism.—A careful study of the relative intake and the excretion of the individual constituents of milk shows us that

(1) Protein digestion and retention are normal.

(2) Fat absorption, according to Freund, is normal. Other substances, which result in the formation of an alkaline reaction within the intestines, will lead to soap stool formation. A substance which does this is casein. This is indirectly shown by the feeding of casein to breast-fed children with dyspeptic stools, which become less frequent and alkaline. The watery stools seen in artificially fed children receiving whey are often quickly improved by adding casein to the diet (Finkelstein); and in well children by adding casein even in moderate quantities an increase in the alkali salts and calcium are seen in the stools.

Fat only in the presence of certain products of food, therefore, leads to the formation of fat soap stools. The presence of fat soap stools is of importance in showing that an excess of fatty acids have been combined with Ca and Mg, but it does not prove that the presence of these stools indicates at all times a pathologic loss of Ca and Mg, as it may only be a relative, and not an absolute, loss. The above illustrates the error of Czerny's conclusions that fats *per se* are the cause of fat soap stools, which Finkelstein claims are due to loss of salt balance. Therefore, the conclusion of Czerny that fat soap stools constitute a symptom of poor fat absorption, with loss of body fat as an end result, is an error. However, an analysis of the stool fat constituents, i. e., neutral fat, fatty acids and soaps, shows an abnormal relation which is of importance. Normally 18 per cent. of the total fat constituent in the stools consists of the insoluble Ca and Mg soaps, of which, in disturbed metabolic balance, approximately 48 per cent. are insoluble Ca and Mg soaps; therefore, it is evident that there is an excessive calcium and magnesium waste through the stools.

Paradox Weight Phenomenon.—Salts other than the calcium and magnesium do not seem to be markedly affected by this negative balance, evidenced by the presence of soap stools. Fat soap stools can only be viewed as of importance in disturbances of nutrition when the Ca balance is pathologically changed. The reversing of the Ca balance results in stationary weight. Nutritional disturbances due to excessive milk feeding are the result of too much earthy alkali being thrown off through the bowel and the organism being robbed of its acid-binding radical, and, therefore, the organism must call on the ammonia radical to neutralize the same. This is evidenced by the presence of the increase of the ammonia coefficient in the urine, i. e., the relation between the ammonia and the total nitrogen products. This phenomenon, discovered by the Czerny school, is especially marked in intoxication and decomposition. The above findings apply to disturbed metabolic balance described by Czerny under the title of *Milch-Nährschaden.* Much less is known of the metabolic changes in the nutritional disturbances due to excessive flour feeding (*Mehl-Nährschaden*). Weigert believed in the latter, that the organism becomes richer in water and salts and poorer in the dry substances. Of the metabolic changes due to infection nothing is known.

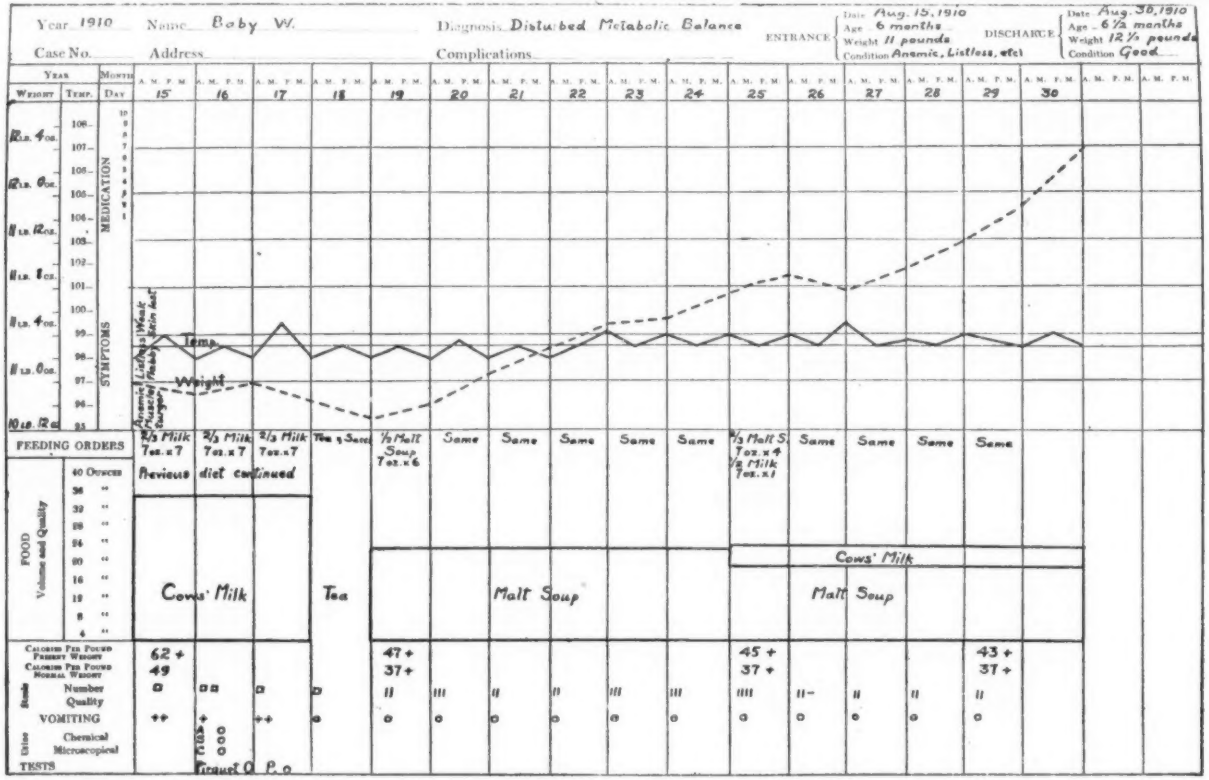

Fig. 1.—Chart showing hospital record of an infant with disturbed metabolic balance.*

*A brief description of the clinical sheet used by me may be of value, as it answers both the needs of a history sheet and daily chart as well. The points illustrated by it are, a graphic relationship between the temperature, weight, quality and quantity of food taken, and the end-results on the stools and urine. Also separate spaces are provided for complications which may influence the preceding under the heading of symptoms, together with spaces for treatment other than dietetic, energy value of foods, vomiting, blood examinations, tuberculin reactions, etc. Gummed slips in six colors, representing different foods used in our wards (human and cow's milk, buttermilk, albumin milk, malt-soup, chymogen-milk) are used to graphically illustrate the quality of food given, the quantity being represented by cross-lines on the colored slips, corresponding with the 4-ounce spaces on the chart. The small figures 1-10 are used to make an electrical reaction curve in cases showing a spasmophilic diathesis.

DIAGNOSIS

The diagnosis of disturbed metabolic balance is based on the history of

1. Food of sufficient caloric value.
2. No gain in weight.
3. Slight variations in temperature.
4. Absence of acute symptoms, but a general loss of turgor.

To condense descriptions of stools and urine, the following characters are used:

STOOLS: | = normal; / = soft; — = quite soft; m = watery; [] = fat soap; x = mucus; c = curds; bl. = Blood.

URINE: A = albumen; S = sugar; Ac. = acetone; D = diazo; I = indican; C = casts; P = pus; Bl = Blood.

PROGNOSIS

The *prognosis* is usually good, even in the artificially fed.

TREATMENT

Change the causative diet. Reduce the quantity of milk and add carbohydrates, or substitute milk low in fats, usually skim- or buttermilk, and add malt or other easily assimilable sugars. In young infants human milk is by far the best form of feeding. The good results of treatment are due to the fact that the tolerance for carbohydrates is not reduced.

1. Diet With Human Milk.—This is by all means the best treatment, especially in young infants. Weight increase may be slow at first, probably due to low salt and protein content of human milk. A loss of more than 6 to 10 ounces over a period of three or four days is frequent. More than this should lead one to suspect an error in diagnosis. This loss may be due, as stated, to stopping of a food rich in proteins and salts, and substituting one low in the same with the continued excretion of water through the kidneys, bowels, etc. This stage is passed in about four days and the system adapts itself to the new ingredients. Other symptoms which must not lead to error are paleness, loss of turgor and sinking cheeks which soon disappear with beginning weight increase. Temperature and pulse do not change, and the stools assume a breast-milk character. If the stage of reparation is slow, and the child does not gain in weight, the substitution of one meal of food rich in protein and salts daily will frequently help (buttermilk or skim-milk). Mother's milk also helps to increase immunity.

2. Diet with Artificial Foods.—In the pathogenesis of feeding of milk, fat plays the most important rôle, and this is best counteracted by replacing it by the well-tolerated carbohydrates. In simple cases reduce the quantity of milk and add carbohydrates, estimating the form of sugar and starches, and if this does not answer, or if the quantity of milk in the previous diet was not too great, then change the kind of sugar. The following answer in most cases: (1) Soxhlet's *Nahrzucker* (nutritional sugar). (2) Cane sugar. (3) Mellin's food. (4) Horlick's malt food.

SEVERER CASES

1. Malt soup (Keller) (laxative), made as follows, is exceedingly valuable:

(1) Milk 11 ounces warmed gradually; add flour 1⅔ ounces; stir constantly, then pour through gauze filter; (2) malt extract (Trommer's, etc., will answer)

3 ounces; boiled water, 22 ounces; potassium carbonate solution, 10 per cent., ⅓ ounce; (3) mix both solutions. Put on slow fire, stir continually, and boil for two or three minutes. Caloric value 800 to the liter. Formulæ: fat, 0.5 per cent.; protein, 2 per cent.; carbohydrate, 12 per cent. This can, at first, be diluted, if indicated. This food should usually be changed after two to four weeks. Loeflund's and Borcherdt's Malt-Soup Extracts do not require the addition of the potassium carbonate solution.

It is indicated in the presence of fat soap stools which soon become pasty and of a mahogany brown color; best between 3 and 6 months; after 6 months add more milk to increase the protein. It is contraindicated below 3 months, if the stools are loose.

2. Buttermilk and skim-milk plus sugar and flour (two carbohydrates). (Antilaxative; action both the same.)

(1) Indicated especially in infants under 3 months.

(2) Valuable over 6 months because of high albumin content.

(3) In convalescence they fill the needs of albumin hunger.

(4) Antilaxative because proteins cause a rich intestinal secretion, which results in putrefaction, which tends to constipation.

(5) Contraindicated in fat soap stools (less valuable than malt-soup in clearing them up).

Human milk diet may cause an early loss of weight because of low salt and protein. Cow's milk diet causes an early, rapid gain in weight due to fixing water in the tissues by increased salts and carbohydrates. Fat rich food causes loss of earthy alkali salts through bowel, resulting in soap building and loss of weight by loss of body salts. This must be counteracted by carbohydrates which increase weight by allowing the organism to use more salts. The early rapid increase is later equalized by the organism and soon becomes less rapid.

General symptoms following change of diet are, better sleep; improved turgor; skin less pale; less variation in temperature. Stools change from soap stools to (1) buttermilk, yellow-brown, alkaline, and fair consistency; (2) malt-soup, acid, softer, mahogany-brown color.

The above changes are due to the protein and carbohydrate constituents.

The results of treatment are due to the fact that the tolerance for carbohydrates is not lessened. Study each case to see if excess of carbohydrates is not being given which is indicated by (a) restlessness; (b) stopping of weight increase after early rise; (c) increase in number, acidity, and amount of mucus in stools; (d) alimentary fever (irregular). If there is irregular fever, human milk must be substituted.

The above mixtures should be gradually replaced by milk-mixtures after two to eight weeks.

OTHER THAN ORDINARY TYPES OF DISTURBED METABOLIC BALANCE

1. The ordinary type, already described (*Milch-Nährschaden*), due to excess of fat.

2. Infections, as otitis, pharyngitis, cystitis, etc. This form readily yields to the same treatment as 1 after cessation of infection.

3. Excessive carbohydrate nourishment (*Mehl-Nährschaden*) is only seen in older children with broader carbohydrate tolerance; in young infants dyspepsia is an early result with excessive carbohydrate feeding.

Treatment consists in administering milk mixtures, i. e., foods high in fat.

II. DYSPEPSIA (STADIUM DYSPEPTICUM OR STAGE DYSPEPSIA)

In practice, especially in young infants, frequently we do not see the stage of disturbed metabolic balance, but see dyspepsia directly, due to an excess of sugar in the food. This can be likened to an excess in whey components when milk is the food used. Fats, unless in excess, probably only play a secondary rôle due to the presence of sugar fermentation. This is characterized clinically by acute gastro-intestinal symptoms, the most marked of which are the stools, which are increased in number and of an abnormal quality. The organism does not show signs of any deep-seated general changes; weight loss is moderate or remains constant, temperature is moderately increased and repair is rapid with the withdrawal of the improper food.

The diagnosis can only be made by a careful consideration of the history, the clinical and functional symptoms, and only that picture of gastro-intestinal findings which developed primarily in a healthy infant or follows directly the stage of disturbed metabolic balance can be diagnosed as dyspepsia and treated as such; otherwise cases of intoxication and decomposition may be neglected.

ETIOLOGY

1. Congenital lowered tolerance to cow's milk.
2. Errors in diet with milk of good quality. (a) Overfeeding with diet of normal proportions (too frequent and too much). (b) Feeding with a diet of improper proportions (excess of sugar, etc.).
3. Feeding with infected milk.
4. Infection of the gastro-intestinal tract (external infections).
5. Systemic infections (otitis, pharyngitis, etc.) associated constantly with a lessened tolerance for food.

SYMPTOMS

Symptoms in General.—Severe symptoms are absent. The mind is clear. The heart action is not rapid. Respirations are not greatly increased. The skin remains unaltered.

Weight.—If there is an excessive sugar feeding in the treatment of a preceding disturbed metabolic balance, we at first get an increase in weight. This ceases later, and dyspepsia develops with moderate weight loss.

The temperature is irregular and moderately increased, and, when it follows diets rich in sugar and salts, is of the alimentary type.

Gastro-Intestinal Symptoms.—The appetite is poor. The mucous membrane of the mouth is red and may be the seat of thrush (due to decreased immunity). Vomiting may be present and usually occurs long after feeding. The stomach may contain food seven to eight hours after feeding, which contains volatile fatty acids (odor), but no free HCl. The abdomen is distended and peristalsis increased (auscultation). Restlessness is marked, probably due to abdominal findings.

Stools.—The clinical diagnosis is usually made from the stools. They are increased in number and watery, and the reaction is usually acid.

The color is green, mucus is present, and curds composed of fatty acids, not casein, are present. The number is more important than the quality, as microscopic examination gives us little etiologic or therapeutical enlightenment, and we cannot get a very definite idea of the quantitative chemical contents.

Bacteria specific for dyspepsia are unknown.

Urine.—Sugar, albumin and casts are absent, unless there is a tendency toward intoxication.

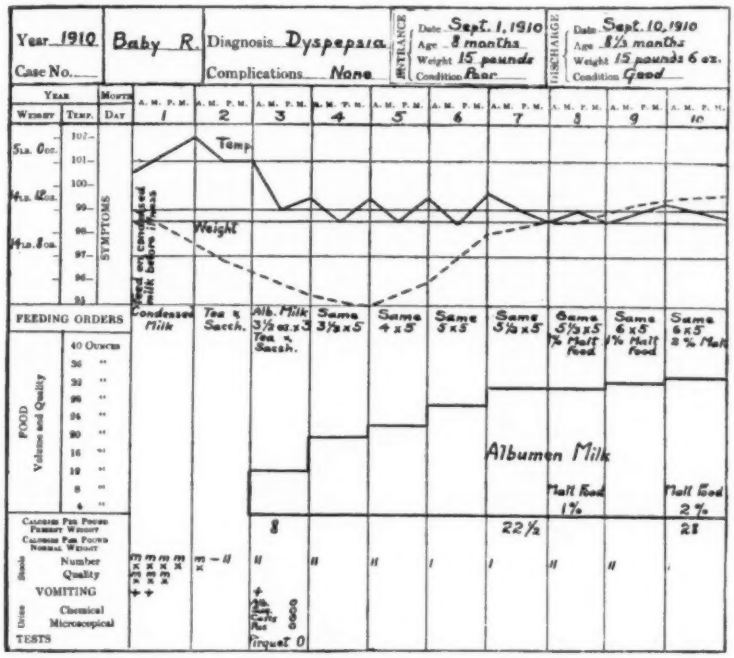

Fig. 2.—Chart showing hospital record of an infant with dyspepsia.

METABOLISM

Changes in part are due to (1) peristalsis; (2) to increased intestinal secretion, which latter accounts for the moderate increase in the protein matter in the stools, and not, as formerly thought, to decreased digestion of the food proteins (casein, etc.). The remainder of the curds is due to fatty-acid salts and bacteria. Fat absorption is usually only slightly reduced. Fat diarrhea is not frequent and only seen in fat-rich foods.

Soaps and neutral fats are in about normal relation to the total fat content. Volatile fatty acids formed from carbohydrates and fats by bacterial action are increased. Sugar is absent. Starch varies directly with the peristalsis. Salts in stools are increased. Green color is usually due to oxidation of bile constituents.

In general, protein retention need not suffer. Ash balance may remain normal. The water in the stools is increased, and in the urine is decreased.

PATHOGENESIS

Sugar and salts, which in the normal child cause no symptoms, in the impaired organism, or in excess, can cause dyspepsia, as evidenced by diarrhea and fever.

Pyrogenic Action of Food Constituents. Action of Salts.—Sodium is the most important salt in raising temperature. Meyer gave a normal child 100 c.c. 10 per cent. NaCl solution per mouth, with usually no result, but in infants with gastro-intestinal symptoms the temperature was raised. Potassium is less important in raising temperature. Calcium rather tends to lower temperature. The mode of action is unknown.

Sugar.—Milk-sugar is more active than malt-sugar in raising temperature, but sugars are less constant experimentally than salts. Whether due to sugar itself or products of fermentation is also unknown. *Proteins* cause no rise in temperature and tend to improve stools. *Fats* have an uncertain and probably indirect action. (They seem to add to the bad effect in the presence of sugar and salt disturbances which, however, cease when the latter are withdrawn.)

The dyspeptic picture is due to (1) slight intestinal epithelial injury following various causes; (2) the administration of certain foods: (a) whey and sugar, which are the most important, and (b) fats, of less importance. These must be administered in quantities large enough to affect the injured epithelium and further impair its function, but not sufficient to cause intoxication.

PROGNOSIS

This is good with proper diet. It is bad with improper diet, with tendency to intoxication and decomposition, varying indirectly with the age of the infant and directly with the previous number of attacks and tendency to recurrence.

TREATMENT

Human Milk.—The younger the infant, the more indication there is for human milk.

Artificial Food.—Remember that primarily the sugar and whey content of the food, and, secondarily, the fats are the irritants.

1. In mild cases reduce the milk to a minimum, and especially the sugar content.

2. In the severer cases reduce the sugar, whey and fats. It is best temporarily to withdraw food entirely as follows:

(1) Starvation (or hunger) for one day (tea and saccharin).

(2) Indifferent diet for second day (low in food value and salts); i. e., Young, one-third milk (best boiled) plus two-thirds thin oatmeal gruel, without sugar. In severe cases, one-third skim milk plus two-thirds thin oatmeal gruel without sugar. Buttermilk may be used in place of milk. The quantity on the second day should not exceed 6 to 8 ounces, as follows: five feedings of 1 to 1½ ounces of food, and 20 to 25 ounces of tea, plus saccharin, given *ad libitum*, giving a total of about one quart of fluids. This will usually answer. Further treatment depends on the reaction to the above; if the general appearance improves, also the disposition, etc., and the weight loss ceases in two or three days (which is the case even in under-feeding), then the treatment was correct, otherwise decomposition must be thought of.

(3) When the weight-curve is constant, the quantity should be increased, but the quality left unchanged; 5×2 ounces, etc., of the same mixture, plus water or

tea to the necessary quantity between the feedings. Weight increase should not be expected because of the low sugar content, but a decrease in weight should always be considered serious.

The stools are, at first, small and contain mucus; later less frequent, and often on milk mixtures without sugar there are fat soap stools which are a good indication.

3. If the temperature and stools are normal after three to five days, carbohydrates can again be added, best in the form of malt preparations (avoid milk-sugar because of its rapid fermentation), beginning with 1 per cent., and gradually increasing to 5 per cent. At first, there is a rapid increase in weight; later a slower one.

Avoid under-feeding too long, even if the stools look bad, if the temperature and weight curves improve, because of the danger of decomposition. Fats can be added in place of sugars, but this should be done with care. (Experiments with cod-liver oil have given me the best results.)

ALBUMIN MILK[1]

The albumin milk of Finkelstein is distinctly indicated in these cases.

Direction for Making: To 1 quart of milk previously boiled for five minutes in a double boiler, add 1 tablespoonful of chymogen (elixir rennet, junket tablets, dessert-spoonful of pegnin or tablespoonful of essence of pepsin). The dish containing the milk is now placed in a water bath, and the milk is heated to 107° F.

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1. Directions for making, recently obtained from the Kinder-Asyl diet kitchen, are as follows: To 1 quart of raw milk add 1 tablespoonful of essence of pepsin (elixir rennet, junket tablets, dessertspoonful of pegnin or chymogen) and stir for one-half minute. The mixture of milk and ferment is now kept at 100 to 107 F. and perfectly quiet for about fifteen minutes, when the clot is formed. The free whey is now poured off and the remaining curd is put into a thin muslin bag, or one made of four layers of gauze (if too thick it drips too slowly or vice versa, curds will pass through and some of the curd is lost), and left to drip for one hour. The curd is now placed in a fine hair sieve and rubbed through with a potato masher, adding buttermilk, one pint, to facilitate this process. The passing through the sieve should be repeated until the curd is minutely divided.

Now, as a final process, water to make a total mixture of 1 quart and 1 per cent. of malt-sugar are added.

This is now put into a receptacle resembling a small ice-cream freezer and put on the stove to boil for ten minutes. While boiling, the handle is vigorously turned, stirring the milk inside thoroughly and constantly (stirring with an egg-beater if the above is not obtainable will answer; the former is to be recommended as it also prevents evaporation. I find that stirring with a spoon does not cause agitation enough to prevent large curd formation.)

Churning is continuously kept up for two reasons: 1. To make the curd finer. 2. To get the suspension more homogeneous. After ten minutes of boiling, the kettle is placed in a bath of cold water to cool it off, still turning the crank for a few minutes until the milk is cool. Then it is put into bottles. Overheating for feeding ruins the product; 100 F. does not change it.

[NOTE: With all due courtesy to the author we wish to state that the first method of preparation was adopted in our diet kitchen, because of the indefiniteness of the details in its preparation as first published and the consequent difficulties encountered in attempting to get a fine curd.]

for thirty to thirty-five minutes. It is now put aside for fifteen minutes, then the excess of whey is poured off and the remaining curd is put into a thin muslin bag, or one made of four layers of gauze (if too thick, it drips too slowly, or if vice versa, curds will pass through and some of the curd is lost), and left to drip for two hours. The curd is now placed in a fine hair sieve, any further free whey drained off and then rubbed through with a potato masher, adding buttermilk 1 pint to facilitate this process. (The buttermilk used is made from whole milk heated to 180 F. for forty minutes, then cooled to 80 F. The scartaline is now added, and after standing from eighteen to twenty hours at the latter temperature it is churned to remove the butter-fat.) The passing through the sieve should be repeated until the curd is minutely divided. Now, as a final process, boiled water to 1 quart is added, and 1 per cent. of malt sugar is added. Sohxlet's Nährzucker is used for my cases, although Mellin's food or Horlick's malt food answers the purpose. Therefore, the finished product contains the curd from 1 quart of boiled milk and 1 pint of boiled buttermilk, 1 pint of boiled water, and 1 per cent. malt sugar.

I have found that rubbing the curd through the sieve with buttermilk prevents the freeing of fat from the curd, so frequently seen when water is used. Boiling the milk, although freeing somewhat less whey, makes only a moderate difference in the quality of the end product, and in our cases has been much better taken by the infants than that made from whole raw milk with its heavier curd which requires shaking during feeding.

Albumin milk prevents abnormal fermentation in the intestinal tract on account of (1) the low content of easily fermentable milk sugar; (2) the low content of whey salts (the latter favor fermentation), and (3) the comparatively high protein content (the latter counteracts fermentation). On standing, a precipitate is likely to form in the bottle containing the albumin milk. Before feeding, the bottle should be well shaken. The quantity to be fed should be warmed to the body temperature and again shaken. Overheating is objectionable. The milk should be kept in a cool place. If thus taken care of, it preserves its quality well, for at least five weeks.

Albumin milk contains 3 per cent. protein, 2.5 per cent. fat, from 1 to 1.5 per cent. of milk sugar, 0.5 per cent. of mineral salts. The mixture contains 450 calories to each liter (Finkelstein). My own tests show the fat content somewhat lower, averaging about 2.2 per cent.

Directions for Use.—Albumin milk is indicated in all infantile disorders which are associated with diarrheas, especially in those cases in which breast milk is not obtainable. It is also valuable in cases of dyspepsia, entero-catarrh, cholera infantum (intoxication, Finkelstein), acute atrophy (decomposition, Finkelstein).

In cases even where the bowel movements continue thin and frequent, or contain mucus, one should proceed to make a daily increase in the quantity of albumin-milk, and after a lapse of eight days begin to add more sugar, or even sooner if the stools warrant it, gradually increasing the dextrin-maltose to a total of 5 per cent. If the weight still remains at a standstill, 2 per cent. of flour may be added. It sometimes happens during the period of convalescence, after improvement has once set in, that an apparently unfavorable condition occurs, characterized by frequent and thin stools. In these cases it may be said with great emphasis that one should not diminish the quantity of the albumin-milk or the sugar, but should continue with the same dosage and quietly wait. After

a short time, if this suggestion is followed, improvement usually sets in; however, when a profuse diarrhea is associated with a marked drop in weight, the sugar must be reduced.

In some of these cases the disturbance is not primarily a digestive one, but it seems to be associated with an infection in some other part of the body. Thus, for example, an intercurrent rhinitis, or bronchitis, will almost invariably cause a standstill or loss in weight, with more or less pronounced digestive disturbance.

Management in Detail.—In cases of dyspepsia, feed the baby tea sweetened with saccharin for six hours. Begin by feeding 300 gm. (10 ounces) of albumin milk, divided into five or six feedings. Tea and saccharin may be given between feedings, as seems indicated. This can be gradually increased by adding 50 to 100 gm. daily until 180 to 200 gm. per kilogram body weight are given. Carbohydrates should be added as previously stated. Children will usually require from four to eight weeks of treatment with albumin-milk. After this time appropriate milk mixtures should be tried. The change from albumin-milk to other mixtures must be made quickly and completely, all bottles being stopped at one time. Relapses are an indication for a return to albumin-milk.

(For further details for its use see intoxication and decomposition.)

Other Treatment.—1. Avoid laxatives (castor oil, calomel, etc.), except for an initial dose. 2. Avoid gastric lavage, at least in early cases. 3. Warm compresses to abdomen aid in removing flatulence, etc. 4. Astringents (tannigen, tannalbin, bismuth subnitrate) are indicated after the weight-curve is steady, temperature normal, and the child generally improved, but the stools still frequent.

III. INTOXICATION (ENTERO-CATARRH; CHOLERA INFANTUM)

This is characterized by violent gastro-intestinal symptoms, rapid loss in weight and other symptoms of deep-seated alterations in the functions of the organism. Collapse and nervous symptoms outweigh the intestinal symptoms. On these findings Finkelstein bases his nomenclature rather than on the intestinal findings, as in previous classifications.

ETIOLOGY

The causative factors are the same as for dyspepsia, i. e., excess of sugar and whey content in the food, but here we have a more severe picture. All factors which cause nutritional disturbances can cause intoxication, but the important point is, which moment in a nutritional disturbance leads to the phase of intoxication?

Factors in a Nutritional Disturbance Which Tend to Intoxication.—*1. Food.* (a) Combination of food elements which individually would be insufficient to cause a disturbance. (b) Infected food, i. e., spoiled milk. *2. Infection.* (a) Gastro-intestinal infection. (b) Systemic infections, as otitis, cystitis, pharyngitis, etc., by disturbing the digestive function. *3. Heat.* Heat can cause a chain of symptoms in itself resembling intoxication. All depends on the individual resistance, a more severe irritant being necessary in the disturbed metabolic balance, and dyspepsia, than in atrophy to develop the intoxication stage.

Carefully following nutritional disturbances teaches us that a definite constituency in the food leads to the various phases, e. g.:

1. Intoxication follows diet rich in sugar and whey content.

2. Decomposition (atrophy) follows diet rich in fat and relatively poor in sugar. These facts are so definite that one can prophesy which variety will prevail in a clinic by knowing the diet commonly in use (Finkelstein).

To develop intoxication there must be an injured intestinal epithelium or preexisting nutritional disturbance. Enteric and systemic infectious processes, as well as heat, give rise to intoxication through their action on the cells of the digestive apparatus in the intestines and the secretory digestive organs. Czerny and Keller believe much trouble is due to infected milk, not to the bacteria themselves, but their acids (toxins formed from fats and sugars by them).

SYMPTOMS

1. High temperature. 2 Rapid loss in weight, even 1 to 2 pounds in a few days, mainly due to loss of water. The skin becomes dry and inelastic. 3. The stools are liquid, are usually numerous and contain mucus and occasionally blood. 4. Collapse. The skin is gray in hue, eyes sunken with distant stare; pinched nose; also frequently incoordinate movements of the extremities, slow and often limited to one extremity; also catatonia of the extremities, and aimless movements, as of chewing, etc. 5. Typical respirations (deep, rapid without pause); described as "toxic respirations." 6. Glycosuria is almost constant, and the sugar is of the same variety as that in food. The phenyl-hydrazin test is the best as the copper sulphate tests require long boiling with lactose, etc., and the reaction may be overlooked. 7. The urine contains albumin and casts. The amount of urine is small, even to anuria. 8. Leukocytosis is present up to 30,000. 9. The heart action is weak, the pulse small and irregular. 10. Vomiting is frequent and the vomitus may contain blood. 11. Nervous symptoms may be pronounced and cause confusion with meningitis. The sensorium is disturbed (with an occasional cry as if in pain). The hydrocephaloid state may be present with strabismus, convulsions, etc. Whether uremia is a factor in this condition is very questionable. 12. Sclerema is constantly seen in the severer types, a very bad sign, due to a coagulation of tissue fluids of an unknown nature (Czerny-Keller).

METABOLISM

Clinical findings lead one to expect marked metabolic changes.

Protein.—Feces.—Nitrogen quota increased, probably due to increased peristalsis and intestinal secretion. Experiments are insufficient.

Urine.—There is a marked increase over the normal under same conditions as to food. During instituted hunger-day the albumin metabolism still progresses and is probably due to toxic processes. Changes from the normal end-products of protein metabolism are present. The ammonia coefficient is increased from 10 to 40 or 50 per cent. (Langstein and Meyer).

1. Change in amino-acids to urea (experiments with glycocoll-amino acetic acid) are interfered with and some excreted as glycocoll in the urine, probably due to a liver insufficiency (disturbance in synthetic production of urea) (Pfaundler). This theory of Pfaundler can also apply to the increased ammonia

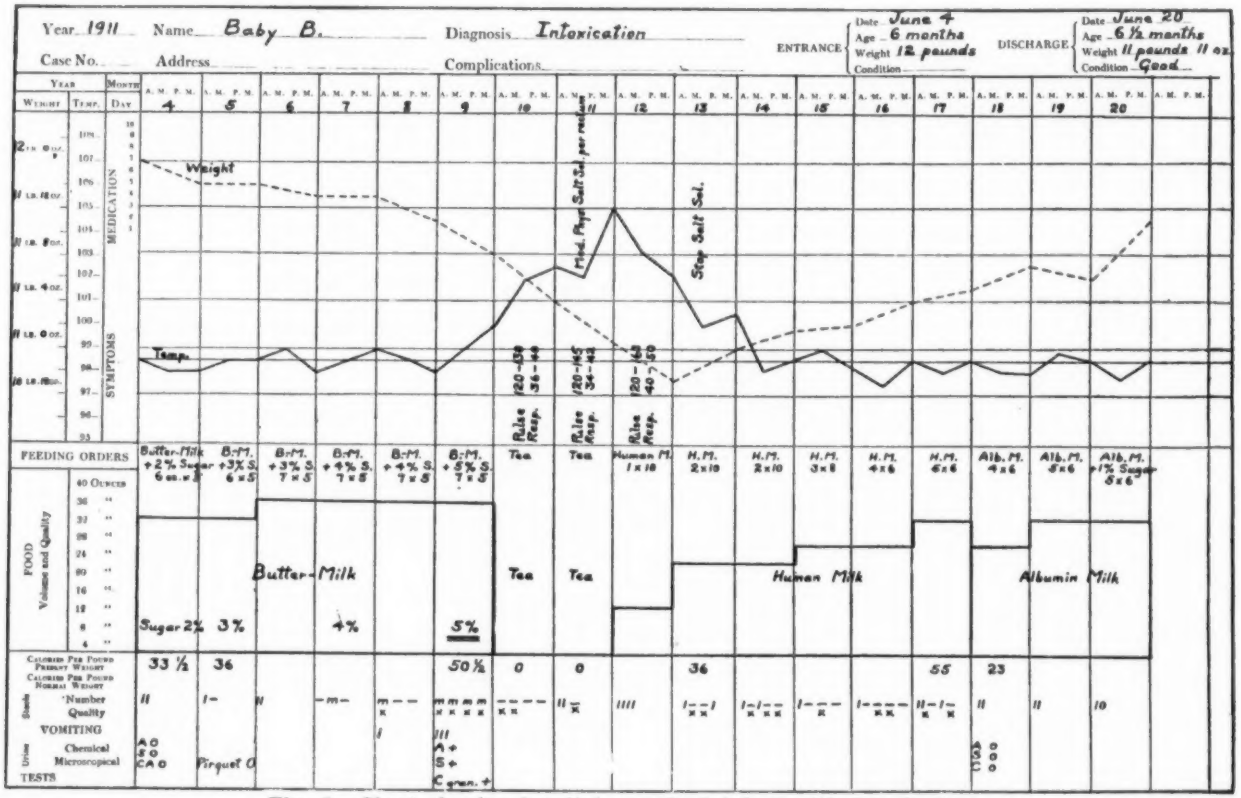

Fig. 3.—Chart showing hospital record of infant with intoxication.

coefficient other factors which probably positively cause increased ammonia coefficiency.

2. Removal through the intestinal tract of Ca and Mg to neutralize the temporary fatty acids, and necessitating the utilizing of ammonia to neutralize the normally formed acids in the urine.

3. The acids (acetone and aceto-acetic acid) formed during the starvation period (toxic) must be neutralized in the urine.

4. Due to interference with carbohydrate metabolism, the fats are not completely burned up in the system by sugar, and as a consequence the acids are combined with alkalies Na and K, and as these are not sufficient the excess is combined with ammonia in place of being thrown out as urea (Rosenstern). This is a typical diabetic reaction (Rosenstern).

Fat.—Meyer found absorption reduced from 97 per cent. to 60 per cent. This improves early in convalescence. Splitting of fat is bad and we get an acidosis with the presence of acetone and aceto-acetic acids (butyric acids, etc., Rosenstern).

Carbohydrate.—Sugars of the type ingested are seen in the urine long before the end of the proper assimilation period. This ceases when the sugar is withdrawn from the diet.

1. With milk sugar in the diet we get milk sugar and galactose in the urine, while other components, as glucose, are missing, on account of the fact that part of the milk sugar is absorbed unchanged, and as it cannot be used as such in the system, it is again excreted, probably due to the injured epithelium allowing it to pass through, and not to the absence of the lactose ferment which Langstein and Steinitz always found present.

The presence of galactose is due to a different cause, and indicates that there is an insufficiency in the sugar oxidation function, and the system is only able to burn up the glucose. This is a liver insufficiency. Other disaccharids have the same ending. Maltose differs from these only in that it can be burned in the system as such without previous splitting in the bowel; therefore, its presence in the urine is double evidence of a lowered power of oxidization by the organism; that is, in its intermediary metabolism.

Water.—Great loss in weight is probably due to the loss of water. Water is lost in the urine and stools which, however, together have been found to equal about the normal amount, only the proportion is inverted, the bowels excreting the greater portion. Loss in body weight is, therefore, in all probability, due to excessive loss (pathologic) through the skin and lungs. Respiratory experiments must prove or repudiate this.

Salts.—(No good experiments to date). Excesses of salts are excreted in the stool, especially the chlorids. There is a negative balance of Cl. It is questionable whether the salt loss is the primary cause of the water loss, or whether the loss of water by the system has its own pathogenesis; indeed, it would require a great salt loss to account for the great loss of water, and it is quite possible that a toxic influence results in a disturbance of the water-binding property.

RELATION OF INTOXICATION TO FOOD ELEMENTS (FINKELSTEIN)

We are able with regularity in dyspepsias to produce intoxication by continuance of the carbohydrate and whey-rich foods; also in light cases

to improve by withdrawing carbohydrates alone; in severer cases by withdrawing both carbohydrates and whey elements.

INTOXICATION IN COURSE OF INFECTIONS

Pathogenesis is not due to a single factor. We know that some are influenced by food withdrawal; others are not. 1. We must believe that the infection injures the intestinal wall and other digestive organs. 2. From this point pathogenesis is the same as in alimentary intoxication. The toxic influences of different infections are as different as are the infections.

PATHOLOGY

In the small intestine there are usually no marked changes. Hyperemia of the mucous membrane and enlarged follicles, especially Peyer's patches, are usually present. Liver, cloudy swelling and fatty degeneration (probable causes of insufficiency). Rott and Tugendreich have believed that an acidity (*Saurung*) of the liver and muscle tissue is present.

DIAGNOSIS

The diagnosis is based on the above symptoms, with improvement on withdrawal of food for twenty-four to forty-eight hours.

Fig. 4.—Infant with intoxication whose hospital record is shown in the chart Fig. 3.

PROGNOSIS

This depends much on reaction to hunger diet, as very severe symptoms disappear often in twenty-four to forty-eight hours. If the same do not disappear in twenty-four to forty-eight hours, in the absence of infection the prognosis is bad. Infections add to the severity. They are bad as a complication of chronic nutritional disturbances.

TREATMENT

1. Removal of all food with sufficient water administration.

2. In severer types, subcutaneous salt infusions twice daily, 100 to 150 c.c., even though there is danger of salt fever in the severer types.

3. Salines per rectum, best administered by the drop method. One drop per second for four hours is 400 c.c. One-half strength of the following solution is good:

	gm. or c.c.
NaCl	7.5
KCl	0.1
CaCl	0.2
Water	1000.0

4. One lavage, if food has been given shortly before.

5. Avoid all laxatives, as the bowels empty themselves; also avoid astringents.

6. Analeptics. Give a mustard bath; reddening of the skin is a good sign.

7. Antipyretics. Use cold packs, and leave the infant undressed.

8. Stimulants. Caffein sodium benzoate, gr. $^1/_{10}$ to ½, four or five times a day; camphorated oil, 1 c.c. every two hours, if indicated; epinephrin solution, 0.6 in 1,000 c.c. of salt infusion; veronal, gr. $^1/_{10}$ per dose, is less depressing than chloral.

9. Sedatives for convulsions, etc.

10. Diet: Hunger diet should be employed not longer than twenty-four to forty-eight hours. Occasional administration of dilute saline solutions (NaCl 5.0, $NaHCO_3$ 5.0, water 1,000), per mouth, also vegetable soups, Moro's carrot soup,[2] Eichel kaffe or thin soya-bean gruel, may soon be used with care, to supplement an occasional tea feeding.

Human Milk.—Human milk is by all means the best food. Feed often and in small amounts, ten times daily, 5 c.c. from bottle or spoon. The first milk should be fat-free. Increase when the temperature, etc., do not react to food, and then not more than 50 c.c. daily.

A sustaining diet should be reached in eight to ten days (32 calories per pound), after which the child can be put to the breast five times daily. If the elevation of temperature returns, except in the presence of infection, cut down the food. The gain in weight is very slow in the stage of repair on human milk, due to the low protein and salt content.

Cow's Milk.—For the first few days after the hunger diet, a food low in fat and sugar should be fed because of the lowered tolerance. One-half skim-milk or buttermilk mixture, without sugar, will answer, but should be fed in small quantities, 10×5 c.c. daily, then 10×10 c.c. On this low diet weight loss and temperature will usually stop. After ten to fourteen days, 32 calories per pound may be fed. Less rapid increase than in human-milk feeding should be the rule. Older infants can be given gruels in their milk. Recurrence of dyspepsia is an indication for return to indifferent food. With a second recurrence breast milk is absolutely indicated. Return to carbohydrates should be made with great care. Albumin milk is indicated in this condition, and the following is a good working rule for its use in these cases: First day, tea. Second day, 10×5 c.c. albumin-milk, with tea *ad libitum*. Then increase 50 c.c. daily until stools are good, then increase 100 c.c. daily until 180 to 200 c.c. is given daily for each kilo weight. After the stools are solid, add 1 per cent. of sugar to the food, and increase gradually to 4 per cent. In intoxication I have obtained better results by at first feeding albumin milk without sugar additions. In infants over 6 months 1 per cent. of flour can also be added. After six to eight weeks, an ordinary milk mixture can be fed. Feedings can now be reduced to five or six daily. Never feed over 1,000 c.c. a day of albumin milk.

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2. *Moro's Carrot Soup.*—One pound carrots are peeled, sliced and cooked one to two hours, and after mashing, poured through a sieve and mixed with the bouillon made from a pound of beef and 1 liter water. To the latter add one teaspoonful of salt.

IV. DECOMPOSITION (ATROPHY: SEVERE TYPE; MILCH-NÄHRSCHADEN: SEVERE TYPE)

Bilanz-Störung may be the direct forerunner of decomposition if the dietetic error is not corrected.

Dyspepsia leads to decomposition if fat predominates in the food and sugar is not present in excess; otherwise we get intoxication. Czerny's internal hunger or cell hunger is the cause of decomposition, in contradistinction to hunger as usually thought of, which is due to lack of food to appease the appetite. It is characterized by general severe changes: (1) Rapid fall in weight; average 20 to 100 gm. daily loss, somewhat slower than in intoxication. (2) Lack of ability to assimilate food. (3) Subnormal temperature with irregular daily curve, with tendency toward collapse. (4) Slow and small pulse, with weak heart-tones. (5) Rapid respirations; prolonged expiration, and irregular breathing, even to the Cheyne-Stokes type. (6) The sensorium is not involved. (7) Urine contains no albumin or sugar. (8) Stools from normal to very bad, depending for their character on the previous diet. Dark brown stools usually indicate duodenal hemorrhages (Helmholtz). (9) The hunger is often very great and cannot be satisfied. (10) Vomiting is frequent

Only a determined effort to limit the diagnosis decomposition to such conditions as are associated with acute rapid loss of body weight (lowered temperature) and general disturbance of the normal body functions will lead us to get good results in our therapeutic applications. It should follow nutritional disturbances in whose pathogenesis alimentary toxins play the chief rôle, and should not be confused with the subacute and chronic atrophies, as tabes mesenterica (tuberculosis), etc.; neither is it to be confused with chronic disturbed metabolic balance, which can be differentiated by a functional diagnosis; that is, a test meal, as described later.

ETIOLOGY

All factors leading to dyspepsia and intoxication may be forerunners of decomposition. At what moment this change takes place can only be answered by the fact that deep-seated organic changes are necessary to its development; these changes which produce such an intolerance toward nourishment may have developed previously to the preceding illness or during its course. Premature infants are especially predisposed, also young infants with previous dietetic errors, and diarrheal attacks; also those fed on one-sided foods, especially carbohydrates, as cereal diets; and also from too long-continued hunger diet. Especially in the young does this apply to cereal waters. All of the preceding reduce the tolerance toward normal food and its assimilation. The tendency to decomposition and, therefore, to narrowing of the nutritional sphere increases with each dyspeptic attack. Death may also be caused by secondary infections, but be unrecognized because of a low temperature, cystitis, etc. (Alimentary sickness, with infectious death, Pfaundler.) Death may also follow quite normal stools without preceding symptoms of dissolution, but this is usually following some dietetic error with cardiac or respiratory failure. This is often the cause of sudden death in our service in malnutrition cases.

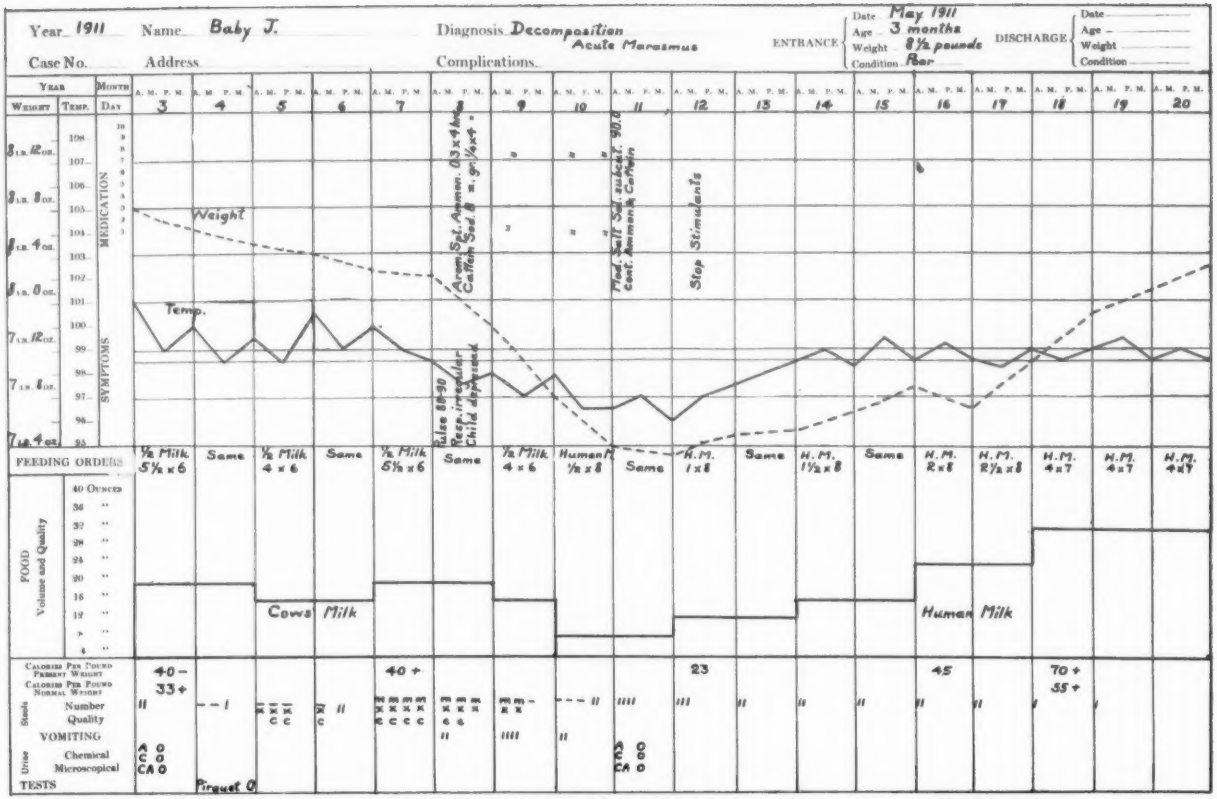

Fig. 5.—Chart showing hospital record of infant in Fig. 6 with decomposition.

DIAGNOSIS

We must ascertain the degree of nutritional disturbance, which is accomplished by noting the history from the present attack backward. If the case be a decomposition, we find an infant who has had a history of repeated diarrheal attacks, with a rapid loss in weight; improvement on hunger diet, with recurrence on refeeding, and a gradual decreasing of the assimilative powers to a point where supportive diet becomes impossible. This difficulty in the stage of repair with proper diet is absent in disturbed metabolic balance and dyspepsia. If this history fails, our only recourse is a very careful test for the reaction to hunger diet and food.

Fig. 6.—Infant with decomposition whose hospital record is given in Fig. 5.

1. *Reaction to hunger diet* is always dangerous. (a) Temperature falls even to collapse. (b) Pulse also slows and weakens. (c) Weight always falls, but varies with the severity of the case. (d) Stools become more nearly normal.

2. *Reaction to food:* This gives a clew to the stage of which Finkelstein recognizes three. The first stage is very much like that of dyspepsia, except that there is a loss in weight with supportive diet. With careful diet the weight loss ceases in from three to six days. In the second stage, with the stopping of food, the weight drop ceases as in the first, but slower; recurrences are likely with the re-administration of food. Rarely can they be saved by the bottle. In the third stage, very little food increases the symptoms. The breast is the only hope. Functional diagnosis with (1) hunger diet, and (2) test meals is therefore equally dangerous.

Weight.—Food administration causes weight drop, and stopping of food is equally as dangerous, a more paradoxical reaction is hard to imagine. It is important to inquire whether shortly before seeing the patient a rich salt and sugar diet had been changed to a poor one, as this might cause symptoms resembling decomposition.

METABOLISM AND PATHOGENESIS

Decomposition is due to mineral loss. In the fat soap stool stage especially, earthy alkalies are lost, and, with diarrhea stools, leads to much more alkali loss. The increased ammonia in the urine is due to the fact that because of alkali losses ammonia is called on to neutralize the acids in the urine. The increased nitrogen waste results from cell destruction following the withdrawal of salts necessary to their vitality. It is a question whether the salt loss is a primary one, or only secondary to the formation of the acids and a call on them to neutralize the acids. The abnormal acid formation in the intestines is the result of bacterial fermentation of the food stuffs (fat and sugar) through bacterial action. This can only occur where the intestinal epithelium has lost its function, or, at least, been impaired. Heubner first pointed this out.

In the condition of the epithelial cells lies the basis of the difference between decomposition and the simple nutritional disturbances, repair of these cells being difficult or impossible in the former, while recovery is rapid in the simple forms. Pfaundler lays the seat of trouble in the intermediary metabolism, and believes that there is a deep-seated involvement of the organ cells throughout the whole organism.

It remains to be proven in how far the loss of salts is the cause of the clinical picture.

1. Loss of alkalies causes loss of water, as they are necessary to the osmotic equilibrium; the water loss through the skin and lungs is very important, as the loss through urine and bowels equalize one another, and the former losses (skin and lungs) probably account for the weight loss.

2. Loss of alkalies causes destruction of cells.

3. Loss of sodium probably accounts for the subnormal temperature.

4. Loss of sodium probably accounts for the slow heart action. Ringer and Loeb have both shown that sodium stimulates and calcium slows the heart's action.

5. Loss of alkalies results in further acidity of the organism (relative acidosis, according to Steinitz; alkalipenie, according to Ffaundler), of whose rule we know as yet very little in relation to the end result.

The healing quality of mother's milk over cow's milk is undoubtedly due, as shown by the experiments of Meyer, to the difference in the whey content, which has a very good influence in the former, and a deleterious one in the latter, on the impaired epithelium.

PROGNOSIS

The prognosis depends on the following:

1. The stage of decomposition.

2. Feeding of human milk. Even light forms offer little hope with artificial foods.

3. Lowered immunity, which adds danger of infection with colds, bronchitis, etc.

THERAPY

Avoid hunger day and feed human milk, about 200 c.c. daily to start.

1. Feeding with Human Milk.—Must be given in moderate quantity, best guarded by drawing off and feeding, as they drink too rapidly (always hungry), and do not stand large amounts. About 200 c.c. daily is enough to sustain the infants temporarily (60 calories per kilo is sustaining, Rosenstern). (*Erhaltungs Nährung*). Feed often; ten feedings may be given, one every two hours (10×20 c.c.); weak tea or saccharin water *ad libitum* between feedings. Expect improvement with the above unless intoxication results from the low salt diet, with resulting loss of water through the stools. If no bad results are seen, increase the food in two or three days. Favorable cases can be fed 100 calories per kilo in ten to fourteen days, and putting to breast may be tried, but the dangers of over-nursing must not be overlooked. Weight may still not improve for some time. (This Keller calls the reparation stage.) This may be shortened by giving one feeding daily of buttermilk or skim-milk, 100 c.c., which is rich in salts and protein. This is not to be done until after the third or fourth week of treatment and with close observation of the result.

Carbohydrates should be added with even greater care. In older children a small amount of farina soup or zwiebach in tea may be tried later.

2. Artificial Feeding.—Change from human to artificial food should always be preceded by a hunger day. After one day of hunger diet small quantities of low fat and sugar diet as buttermilk or skim milk without flour or sugar, or, if not at hand, whey whole or diluted, may be given, using 10×10 c.c., and with it saccharin water or tea. Increases must be more careful than with human milk. If results are good, we may increase in two weeks to 70 calories. Add carbohydrates and fats with great care. Carbohydrates increase weight more than fats. Maltose is less likely to affect the bowels and is more assimilable. One per cent. may be added at first, increasing very slowly to 5 per cent. In infants older than 3 months, in addition flour may be used. Always expect a setback with new food additions.

3. Albumin Milk is of value in decomposition where human milk is not available; however, my results would lead me to make every effort in the future to feed this class of infants with human milk. Stimulants, artificial heat, and, in severer types, transfusions are indicated.

V. MIXED FORMS OF NUTRITIONAL DISTURBANCES

Bilanz-Störung (disturbed metabolic balance) is frequently associated with dyspeptic symptoms. Again, these may show signs of intoxication. Decomposition is the form most subject to complication.

THERAPY

Disturbed metabolic balance (*Bilanz-Störung*) with dyspeptic symptoms, treat as a dyspepsia by reducing diet. In dyspepsias with signs of intoxication, employ hunger diet, etc.

Decomposition complicated by intoxication is the severest combination, but can be recognized, if careful consideration is given to the history and the infant's general condition. The greatest danger lies in the fact

that in the former we must nourish; in the latter starve. Without a hunger day either human or albumin milk should be given in small quantities, 10×5 c.c., and increased 50 c.c. daily under control of the clinical symptoms. After eight to ten days of the above the infant can usually be put to the breast five times daily. The weight may remain stationary, and one must judge by the stools and general condition as to the addition of further foods. With albumin milk, after three or four days, the weight becomes stationary and the stools of fat soap character; then gradually some malt sugar can be added.

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METABOLISM AND NUTRITION

Importance of Proper Balance of Fat and Lime in Infant Feeding

(K. Stolte: Jahrb. f. Kinderh., 1911, lxxiv, No. 4, p. 367)

Stolte comments on the conflicting statements of various pediatrists in regard to infant feeding, some obtaining excellent results with one food-mixture, while others have found this mixture harmful; some denouncing the very elements in the food to which others ascribe its efficiency. Looking over the entire field from a higher standpoint shows, he says, that one feature is common to all the cases in which the infants thrived, namely, that the stools became less fluid. His research indicates that this change in the consistency of the feces is the result of soap production and absence of fermentation. The aim in feeding, therefore, should be to insure soap production and avoid fermentations. From this standpoint the benefit from the most varied systems of infant feeding is easily understood; each by different routes arrives at the same goal. The essential point is the proper balance of fat and lime in the bowel to insure soap production. If too much or too little of either is present, the desired end is not attained. Fermentations induce the production of fatty acids, which irritate the intestines and interfere with saponification. These views explain the advantages of albumin-milk, as the intestinal flora with this induce putrefaction rather than fermentation. They explain further the tolerance of carbohydrates with buttermilk, which contains especially large proportions of lactic acid and albumin. They also explain how diarrhea may be arrested by albumin-milk, by buttermilk, by diluted milk, or by milk and lime-water. With cow's milk more lime is needed to saponify the fatty acids generated in the intestine by splitting of the fat. Stolte found it possible to check diarrhea almost unfailingly when he gave the infants lime-water with the diluted milk, without carbohydrates. The more complex the structure of the carbohydrate given, the longer it takes to be broken down into its ultimate elements, and thus the less material to ferment in a given period of time, and hence the less opportunity for the fermentation producers to get in their work. These more complex polysaccharids include dextrin, flours and malt. This difference between the various carbohydrates was instructively shown in one of his cases in which the infant had diarrhea on albumin-milk and cane-sugar, while the stools became formed when flour was substituted for the sugar, and the stools became actually hard when nothing but a cereal gruel was given with the albumin-milk. It is possible that the property of promoting putrefaction rather than fermentation is the chief advantage of the albumin-milk. Infants thrive on breast milk after serious illness, although breast milk is poor in albumin, showing that the protein content alone is not the essential factor.

W. T. C.

ACUTE INFECTIOUS DISEASES

Acute Rheumatism Among Children

(*F. Langmead: Lancet, London, 1911, clxxxi, 1133*)

Altogether 2,556 children were examined by Langmead. Of these, 844 were boys and 838 girls in the senior departments, i. e., from 6½ to 14 years old; 874 were attending the infants' department, and consisted of nearly equal numbers of boys and girls from 3 to 6½ years old. Fifty-nine boys and 74 girls could be classified as definitely rheumatic, a total of 133. This is equivalent to a percentage incidence of 5.20 for children of all ages attending school, or of 6.83 for children in the senior departments only, those attending the infants' department being excluded from the estimation. In the majority of cases attention was drawn to the rheumatic child by the condition of the heart, sometimes by definite valvular disease, at others by dilatation accompanied by such alteration in sounds and rhythm as one is accustomed to hear in rheumatic children. More and more children become rheumatic as they grow older. There is, however, an apparent accession of rheumatism at the age of 10 which is not shown in subsequent years. Langmead gives no explanation for this and regards it as an inaccuracy which would have disappeared if a large enough number of children had been examined.

Out of these 133 children all except eighteen showed some evidence of cardiac disorder at the time of examination. Thus rheumatic heart affections occurred in 4.49 per cent. of the children of all ages attending school, or in 5.94 per cent. of those in the senior departments only. Obvious valvular disease was found in 2.93 per cent. of children at all ages, or 3.92 per cent. of those in the senior departments. Mitral regurgitation was noted forty-four times; aortic regurgitation and mitral stenosis only once; dilatation with a systolic bruit at the apex, possibly mitral regurgitation, twelve times; dilatation with a short presystolic rumble, or so-called reduplicated second sound (mid-diastolic bruit), possibly early mitral stenosis, eight times; dilatation without bruits, twenty times. The comparative rarity of aortic regurgitation in children is emphasized by the fact that only one case was discovered among the 2,556 children examined. Chorea did not appear among the children or in the histories of their previous illnesses as often as might have been expected. There were eighteen cases only, which amounted to 0.70 per cent. of the children of all school ages or 1.06 per cent. of the children in the senior departments. The comparative incidence in boys and girls followed the usual rule, there being twice as many girls affected as boys. Thus 0.71 per cent. of the boys were at some time choreic and 1.43 per cent. of the girls. The relation of enlarged tonsils and adenoid vegetations to rheumatism in childhood is well illustrated by the figures obtained. Taking the 133 rheumatic cases, in seventy-five there were no signs of abnormality in the throat, in twenty-one the tonsils were slightly enlarged, in eleven considerably enlarged, in sixteen greatly enlarged and in three tonsillotomy had been performed before the patients were examined. Adnoids were responsible for producing signs of post-nasal obstruction and mouth-breathing in seven. In other words, there was an abnormality of the tonsils or pharyngeal mucosa or both in fifty-eight cases, or in 43.6 per cent. of the rheumatic cases. This was sufficient to warrant operative interference in thirty-seven cases, or 27.8 per cent. of the rheumatic cases.

F. C. ZAPFFE.

Diphtheritic Paralysis

(*Andrew Love: Glasgow Med. Jour., 1911, x, 245*)

The cases on which this article is based were under Love's care in Belvidere Fever Hospital, Glasgow, during the years 1900-1906, inclusive, and in Gateside Fever Hospital, Greenock, during the years 1908-1911, inclusive. The number of cases of diphtheria admitted during the period selected was 1,313, and of these eighty-five or roughly 6 per cent. became paralyzed. The years 1905 and 1906, and more especially 1906, show an enormous increase in the number of cases of

paralysis. This was principally due to the greater severity of the disease during these years. Of the total number of cases observed who suffered from paralysis, thirty-four were males and fifty-one females, their ages ranging between 14 months and 40 years. Paralysis was more common at those ages when the incidence of the disease is greatest. Generally speaking, the later the day of illness before antitoxin is administered the greater is the toxemia, and naturally, the greater the danger of paralysis supervening. Of these eighty-five patients, sixty-five, or 77 per cent., recovered, and twenty, or 23 per cent., died. In no one case could death be attributed solely or directly to paralysis, although in all cases it was contributory. In ten of the cases that terminated fatally the disease was of the malignant type, while in the others it was of a severe character. Eight had nasal implication, seven a combination of faucial and laryngeal diphtheria with tracheotomy in three instances, and twenty-seven had greatly enlarged cervical glands. Of the latter, two went on to suppuration. Of the eighty-five cases, fifty-three, or almost two-thirds, had paralysis of the palate alone; fifteen of the eyes and palate; three of the eyes alone (strabismus). Of the fifteen who were afflicted with paralysis of the eyes and palate combined, three had paralysis of accommodation as well as strabismus. Paralysis of the ocular muscles alone is uncommon, and only two of the sixty-eight patients recorded were affected in that way. Of the twelve patients noted as having intercostal and cardiac paralysis, nine died and three recovered, the main symptoms being great difficulty in inspiration with marked feebleness and irregularity of the heart. In the one case in which the lower limbs only were affected the loss of power lasted only for a few days. The average duration of the paralysis was eighteen days. Two of the cases had paralysis of the palate on admission to the hospital, and the condition persisted until death occurred. In the cases in which death occurred the average number of days of residence in hospital was twenty-one, while the average number of days spent in hospital by those who recovered was 70.5. It is interesting also to note the persistence of the membrane on the throat after the administration of antitoxin. In no instance did the membrane disappear before the third day after admission to hospital, and only then in the milder cases. The average number of days that elapsed in the cases here recorded before the throat was free from membrane was 7.2; the longest period noted during which the membrane persisted was fourteen days. Vomiting in diphtheria is always regarded with apprehension, as it is frequently associated with cardiac failure. In the cases here recorded vomiting occurred in fourteen, and nine of these had a fatal termination. It is noteworthy that the vomiting occurred simultaneously with the onset of the paralysis. The urine varied greatly as regards albumin. Of the eighty-five cases, twenty-seven had a large quantity of albumin in the urine, eighteen a moderate quantity, seventeen a mere trace, and twenty-three no albumin at all. With one exception there was a large quantity of albumin in the urine of all the fatal cases. In the cases under consideration fifty-six received a single dose of antitoxin, the smallest being 6,000 units and the largest 75,000 units, while the average amount administered was 17,000 units. Second doses were given in twenty-four cases, the average in these being 17,500 units. In five cases only were third doses resorted to, the average in these instances being 9,500 units. The over-all average, that is, the average aimed at by taking all the doses administered to all the patients, is represented by the somewhat large amount of 22,000 units. In connection with the administration of antitoxin, it is interesting to note that out of the eighty-five cases dealt with, thirty-eight were afflicted with serum rashes. In twenty-eight cases the rash was urticarial in character and general in distribution; in six it was scarlatiniform, and in four morbilliform. Two of the rashes, which on their first appearance were urticarial, became morbilliform in about twenty-four hours, while one, which was in the first instance scarlatiniform, became morbilliform in the same time. The serum rash when urticarial in character appeared usually on the ninth day after the administration of serum, and lasted on an average 4.2 days. The scarlatiniform rash also appeared on the ninth day, but usually lasted only two days,

while the morbilliform rash came out on the twelfth day after the injection of serum, and as a rule lasted four days. All important as is the early administration of antitoxin, it is a mistake to suppose that in the late cases antitoxin is of no avail. Twenty-seven of the patients were admitted after the fifth day, and all received antitoxin. Of these, five died, a mortality of 18.5 per cent.

F. C. ZAPFFE.

Blood-Pressure in Diphtheria

(*J. D. Rolleston: Brit. Jour. Child. Dis., 1911, x, 433*)

This paper is based on observations made on 179 cases of diphtheria admitted to the Grove Hospital from July, 1909, to December, 1910. The blood-pressure in each case was taken with C. J. Martin's modification of Riva-Rocci's sphygmomanometer. The systolic pressure, as measured by disappearance of the radial pulse, was alone taken into consideration. As far as possible the measurements were taken at the same time each day between 10:30 a. m. and noon; i. e., from one and one-half to five hours after food; in many cases an evening record between 6:30 p. m. and 7 p. m. was also made. The blood-pressure was taken daily in each case until the patient was allowed to sit up; i. e., for a period ranging from three weeks in the mild cases to six or eight weeks in the severe. Comparative observations were subsequently made on the different readings in the recumbent and erect positions. Apart from the early fatal cases the total period of observation in each case ranged from four to fourteen weeks. All the patients, except fifteen who were above the age of 15 years, were children. Ninety were males, eighty-nine were females. There was no appreciable difference in the blood-pressure of any two individuals of different sexes but of the same age and suffering from attacks of diphtheria of equal severity. Of the 179 cases, sixty-three, or 35.1 per cent., according to Cook and Brigg's estimate, and forty-five cases, or 25.1 per cent., as judged by Seiler's standard, showed for varying periods a pressure inferior to the normal. Owing to the well-known depressive effects of diphtheria toxins these figures appear surprisingly low, but are explained by the inclusion of a large number of mild cases, in which the blood-pressure was not lowered in the course of their illness. The varying degrees of depression bore a direct relation to the severity of the attack, being pronounced and persistent in the severe and slight and transitory in the mild cases. Of those whose blood-pressure was not lowered, thirty-six cases, 20.1 per cent., according to Seiler's standard, showed a reading which always exceeded the normal. Judged, however, by the more liberal estimate of Cook and Briggs, the pressure of these cases did not persistently rise above the normal limits. In ninety-seven of the 130 cases admitted in the first week of disease and in ten of the fifteen cases admitted in the second week, the highest reading was the first taken. The occurrence of the comparatively high pressures in the first week is attributed partly to the febrile disturbance of the acute stage before the diphtheria toxins had had time to produce their characteristic effect and partly to the psychical action caused by the application of an unfamiliar instrument, the latter fact accounting for the highest reading being usually obtained on the first measurement. The preponderance of the lowest readings in the second week accords with the clinical fact that this is the time of predilection for the so-called "cardiac paralysis," which should more accurately be called vasomotor paralysis, the heart failure being secondary to the vasomotor affection. Apart from one case of diaphragmatic paralysis, all the deaths in the present series were due to this cause. The lowest reading in a patient who recovered was 60 mm., which was registered on one occasion in a girl aged 3 years. In only ten other patients who recovered did the pressure fall below 80 mm. in patients above the age of 2 years. The pressure in forty cases returned to the normal by the end of the seventh week. Forty-two cases, or 23.4 per cent., showed various degrees of laryngeal involvement. In thirteen severe cases tracheotomy was performed; in eleven moderate cases the symptoms, though fairly well marked, did not require surgical interference; in eighteen mild cases the laryngeal symptoms consisted merely of a croupy cough and husky

voice, thus representing the first degree of croup of earlier writers. The effect of laryngeal obstruction on the blood-pressure was most marked in the severe cases. Increase in dyspnea was followed by a rise of pressure and the relief of the obstruction on opening the trachea was immediately followed by a considerable fall. The rise of blood-pressure in the moderate laryngeal cases was less than in the severe. The blood-pressure in the mild laryngeal cases during the acute stage was not any higher than in the purely faucial cases; in fact, it may be said to have been dominated by the character of the concomitant faucial attack. In the great majority of early serum rashes, erythematous or urticarial, no change occurred in the pressure, while in a large percentage of late eruptions, which are usually associated with febrile disturbance and often with pains in the joints, muscles and fasciæ, the pressure was raised. Thus of seventy-seven cases at the time of the early eruption, sixty-two patients, or 80.5 per cent., showed no change in the pressure, in nine it was raised and in six it was lowered. On the other hand, out of fifty with late rashes, in twenty cases, or 40 per cent., the pressure was raised, in twenty-eight, or 56 per cent., there was no change, and in only two cases, or 4 per cent., it was lowered. The rarity of any rise in pressure at the time of the early rash is in keeping with the fact that during the second week of the disease, in which most of these rashes occur, the pressure is usually at its lowest. In the third week, in the course of which the late serum phenomena occur, the pressure as a rule is no longer falling, while on the other hand the fever and joint pains tend to raise it. As arterial hypotension and albuminuria are both phenomena of diphtheritic intoxication, any change in the blood-pressure coexistent with the occurrence of albuminuria was almost invariably in a downward direction. Thus among fifty-one cases in whom albuminuria was not present on admission, its onset was accompanied by a fall in the pressure in twenty-six and in twenty-five there was no change; in another eight cases increase of preexistent albuminuria was accompanied by a fall in the pressure. The solitary exception to the rule occurred in a girl aged 6 years, in whom severe faucial diphtheria was complicated on the twenty-ninth day by uremic convulsions, one of the rarest events in diphtheria. On the two following days the blood-pressure, which up till then had not been examined, stood at the disproportionately high figure of 120 mm., falling on the thirty-second day to 80 mm. Death took place on the thirty-third day. Albuminuria in diphtheria thus offers a striking contrast to scarlatinal albuminuria, during which it is usual for the blood-pressure to be raised. Thus in eleven such cases Rolleston found a rise in all but two. In early paralysis the blood-pressure tended to fall. In late paralysis, even when extensive, it was usually not affected. Rolleston concludes that sphygmomanometry in diphtheria, as in other acute diseases, though of considerable theoretical interest, has little practical significance.

F. C. ZAPFFE.

TUBERCULOSIS AND SYPHILIS

Consumption and Order of Birth

(W. C. Rivers: Lancet, London, 1911, clxxxi, 987)

Rivers observed in patients' case-sheets which showed ordinal position in the family, a large number of entries of eldest-born children, and from this observation conceived the idea that clinical pulmonary tuberculosis might affect such persons more than others. Brehmer's and Riffel's reports show that both first- and second-born offspring appear subject to consumption at a very much higher rate than younger members of a family. Rivers' own observations in 263 cases confirm those of other clinicians. Mitchell, among 443 idiots and imbeciles, found 138 first-born, or 31.1 per cent. Langdon Down claims liability to idiocy among primiparous children because 24 per cent. of the 2,000 idiots (illegitimate largely excluded) examined by him were first-born children. Among Wilbar's 675 there were 191 first-born; among 100 of Beach's, twenty. Thus, respecting

the exact amount of increased risk of developing phthisis that an eldest child runs, no direct data exist from which to calculate. As matters stand, one may say that whereas in 1,228 consumptives (494 upper middle-class Germans; 353 middle- and lower-class inhabitants of Lancashire, London, and Northumberland, and 381 mainly lower-class Lancastrians) one should find 196 eldest children, one actually does find 281, i. e., eldest-born consumptives are in excess, say, 40 per cent.

Of 141 of Rivers' last cases there is one patient with diabetes mellitus, three who suffer from hay fever, and three the subjects of congenital xerodermia. The diabetes patient is an eldest child; two of the hay fever patients are likewise, while the remaining one is second-born, and two of the ichthyotics are eldest-born. Yet the first- and second-born of these 141 only number fifty-eight and the first-born alone only twenty-eight. This, in Rivers' opinion, would seem to point to the conclusion that the tendency of the eldest-born to become consumptive is an innate tendency and the hypothesis is strengthened, he says, if we appeal to the literature of the subject. Lunatics and idiots are certainly very liable to consumption, and, as has been seen, it is exceedingly likely that they are disproportionately often eldest-born. Again, if faulty upbringing by inexperienced parents is to be made responsible for elder-born criminals, lunatics, idiots and perhaps men of genius, this would render ordinary human responsibility sufficiently formidable. The existing evidence negatives the view that infection is the sole cause.

F. C. ZAPFFE.

TRANSMISSION OF DISEASE BY MEANS OF BOOKS

Request for Data

I am preparing a paper on "Books as a Source of Disease," to be read before the next International Congress on Hygiene, and in order to obtain data, I respectfully request the readers of this note to send me an account of any cases, the sources of which have been traced to books or papers, or where the evidence seemed to make books or papers the offender. I further request information concerning cases in which illness or even death has been caused by the poisons used in book-making.

All the information possible is wanted in order to present as complete a paper as possible. As in the case of insects which we now know to be "carriers of disease," it is first necessary to collect the scattered evidence in order to show that there is real danger in books; and this will compel better care to be taken of libraries and books and improve the health of mankind.

The Free Library of Philadelphia, Department of Public Documents.

WILLIAM R. REINICK,
1709 Wallace Street, Philadelphia.

BOOK REVIEW

Case Histories in Pediatrics. A Collection of Histories of Actual Patients Selected to Illustrate the Diagnosis, Prognosis and Treatment of the Most Important Diseases of Infancy and Childhood. By John Lovett Morse, A.M., M.D., Assistant Professor of Pediatrics, Harvard Medical School; Associate Visiting Physician Infant's Hospital and Children's Hospital, Boston. W. M. Leonard, Boston, Mass., 1911.

The title of this volume describes its purpose and mission. Teaching medical students by means of actual case histories originated at Harvard several years ago. The author states in his preface that this method of teaching is far superior to recitations, quizzes and conferences. It compels the student to think for himself. It is almost as valuable as the clinical lecture, in which the patient is shown, and is more instructive than the didactic lecture.

This book should inspire every physician who teaches any branch of clinical medicine to follow out the same method with his own cases. The description of

each case is so clear and concise and the essential points in the examination and diagnosis so forcibly brought out that this little volume could well serve as a pattern.

There are one hundred cases cited, which are grouped into Diseases of the New-Born, Gastro-Intestinal Tract, Nutrition, Infectious Diseases, etc. Each case illustrates a different disease, except in a few instances, wherein two examples which illustrate different phases of the same disease are included.

The value of this book would be greatly enhanced if more illustrative cases were included under certain sections. It is impossible to give a comprehensive picture of diseases such as tuberculous meningitis, bronchopneumonia, etc., by quoting one case.

The omission of cases illustrating chorea, tuberculosis, asthma, enuresis and the acute exanthemata, which are important and characteristic diseases of childhood, should be remedied in later editions.

The discussion of the differential diagnosis is full of practical points which the author has gleaned from his own wide personal experience. The treatment is based on what was needed and undoubtedly advised in each individual case.

There is nothing in this volume that savors of the text-book. The general practitioner will find many valuable suggestions and useful hints which are not found in other books. The index is complete and reference to the various diseases can readily be made.

The book would be more readable and satisfactory from a typographical point of view if the name of the disease were printed prominently as a heading to each case.

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